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ELECTRICAL SKIN RESISTANCE TEST IN EVALUATION OF PERIPHERAL NERVE INJURIES

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THE MEASUREMENT of electrical skin resistance has been recommended by Richter and Katz¹ and Jasper and Robb² as a practical test in the evaluation of injuries of peripheral nerves. This method, in contrast to the sensory examination, does not depend on the cooperation of the patient. It can therefore be used with uncooperative, or even unconscious, patients and may give objective results in cases of hysteria or suspected malingering. Richter and Katz examined 10 patients with injury of the ulnar nerve and found a correlation of skin resistance with sensory changes in most of them. Of the 27 patients with various peripheral nerve lesions studied by Jasper and Robb, all but 2 showed a correlation of the areas of increased skin resistance with the areas of sensory loss. This method is now being used on a large scale in the evaluation of nerve injuries and in the study of recovery

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1. Richter, C. P., and Katz, D. T.: Peripheral Nerve Injuries Determined by the Electrical Skin Resistance Method, *J. A. M. A.* **122**:648 (July 3) 1943.

2. Jasper, H., and Robb, P.: Studies of Electrical Skin Resistance in Peripheral Nerve Lesions, *J. Neurosurg.* **2**:261 (July) 1945.

after various methods of suture (White and Hamlin³; United States War Department⁴). It is felt that the return of an increased skin resistance to normality is an indication of recovery of autonomic function, primarily that of innervation of the sweat glands (Richter, Jasper and Robb).

During the past two years, a large series of peripheral nerve injuries has been studied at the Neurological Institute of New York by a group of investigators working in collaboration with the United States Naval Hospital, St. Albans, N. Y. The extent of the injury has been determined and the course of recovery, both spontaneous and after nerve suture, has been followed. The objective was the evaluation of practical methods of studying motor, sensory and autonomic functions. In cases of complete and partial denervation and during the course of reinnervation, the extent of reduction of motor function, as defined by a thorough clinical examination, analysis of a moving picture film and chronaxia studies; of sensory function, as defined by clinical examination, and of autonomic function, as defined by electrical skin resistance, has been studied and correlated.

This paper presents the results of the investigation of the electrical skin resistance test as a practical means of evaluating dysfunction of peripheral nerves.

METHODS OF INVESTIGATION

The electrical skin resistance was determined with the improved Dermohmmeter of Jasper.⁵ The technic used was that described by Richter and his associates⁶ and by Jasper⁵ and Jasper and Robb.² Quantitative measurements were not taken regularly, as it proved that the gradient of electrical skin resistance between normal and impaired areas was usually great and the boundaries quite distinct. After the area of sensory change was outlined, the electrical skin resistance of the normal areas was determined (basic resistance). The area with a change in resistance was then outlined. The instrument has a very wide range of resistance values, and the variable resistor could always be adjusted so as to obtain a large scale deflection of the meter in the normal area. In this way significant differences could be seen when they existed. Finally, comparison was made always with corresponding areas of the other side. The normal pattern of variation in low and high electrical skin resistance of the hands and feet, as mapped out by Richter, Woodruff and Eaton,⁷ was also considered carefully.

3. White, J. C., and Hamlin, H.: New Uses of Tantalum in Nerve Suture, Control of Neuroma Formation, and Prevention of Regeneration After Thoracic Sympathectomy, *J. Neurosurg.* **2**:402 (Sept.) 1945.

4. Neurological Diagnostic Techniques, United States War Department, Technical Bulletin (TB Med. 76), Washington, D. C., Government Printing Office, 1944.

5. Jasper, H.: An Improved Clinical Dermohmmeter, *J. Neurosurg.* **2**:257 (July) 1945.

6. Richter, C. P., and Woodruff, B. G.: Changes Produced by Sympathectomy in the Electrical Resistance of the Skin, *Surgery* **10**:957 (Dec.) 1941. Whelan,

We found that the normal sweating of the subject was usually quite adequate to make possible differentiation between normal and denervated areas when the patient was examined in a fairly warm room. On rare occasions only Furmethide (furfuryl trimethyl ammonium iodide), 2.5 to 5 mg., given subcutaneously, was used to induce sweating and thus to lower a high basic resistance that made comparisons difficult and faulty. It is felt that this drug stimulates sweat glands with intact nerve supply, but not denervated glands (Guttman⁸).

Sensory examination consisted in the testing of tactile sensibility and pain sensations due to pinprick, with the occasional addition of two point discrimination, localization of pain and touch sense and deep pain sensation.

Examination of the motor function in this particular correlative study consisted in the usual clinical observation, supplemented with determinations of chronaxia.

Our material consisted primarily of 87 cases of injuries of the following peripheral nerves: ulnar, median, radial, sciatic, peroneal and tibial. Fifteen cases of injury of the brachial plexus were also studied. In all cases the lesion was distal to the point of junction of the sympathetic fibers with the nerve.

The interval between injury and examination varied, since most patients were brought from overseas. However, many cases of complete nerve section could be studied directly after operative procedures, especially that of "plasma glue suture" (Tarlov⁹).

Although many patients were examined several times in order to follow the course of improvement and the rate of regeneration, the time element involved in this slow process does not permit us to outline the results of testing during the whole course, from injury to complete recovery. However, the results in cases of complete lesions of nerves and in cases of lesions of varying incompleteness can demonstrate the practical value of any test in following a course of regeneration.

In the material to follow, the results of the investigation of motor function, the sensory examination and the skin resistance test are correlated.

Motor function is classified as follows: (1) complete paralysis and complete neuromuscular degeneration; (2) definite drop in chronaxia as compared with the values obtained in a former examination, but no response of muscles to stimulation of the nerve; (3) partial neuromuscular degeneration, with some muscles responding to stimulation through the nerve.

The results of the sensory examination are simplified and classified as anesthesia, hypesthesia and hyperesthesia. Emphasis is placed on the correlation between the electrical skin resistance and the sensory function. Varying degrees of sensory loss in the total sensory area of the nerve are also considered in this correlation.

MATERIAL

Injury to the Ulnar Nerve.—In 10 of the 20 cases of injury to the ulnar nerve the total area supplied by this nerve was anesthetic. In

F. G., and Richter, C. P.: Electrical Skin Resistance Technic Used to Map Areas of Skin Affected by Sympathectomy and by Other Surgical or Functional Factors, *Arch. Neurol. & Psychiat.* **49**:454 (March) 1943. Richter and Katz.¹

7. Richter, C. P.; Woodruff, B. G., and Eaton, B. C.: Hand and Foot Patterns of Low Electrical Skin Resistance: Their Anatomical and Neurological Significance, *J. Neurophysiol.* **6**:417 (Sept.-Nov.) 1943.

8. Guttman, S. A.: Use of Furmethide in Testing Sweat Secretion in Man, *Arch. Neurol. & Psychiat.* **51**:568 (June) 1944.

9. Tarlov, I. M.: Autologous Plasma Clot Suture of Nerves: Its Use in Clinical Surgery, *J. A. M. A.* **126**:741 (Nov. 18) 1944.

7 of these cases there was complete neuromuscular degeneration, recovery being in the beginning phase in the remaining 3 cases. In all but 1 of these cases with complete paralysis and sensory loss there was a definite increase of electrical skin resistance in an area which corresponded fairly well to the area of sensory loss. In the 1 case no change in electrical skin resistance was found.

In 5 cases hypesthesia of the total ulnar area was present. In all these cases signs of motor recovery were apparent. In 3 cases there was a correlating area of increased electrical skin resistance. In 2 cases no change of electrical skin resistance in the ulnar area as compared with surrounding areas could be found, but in 1 case the resistance of the whole affected hand (ulnar, median and radial areas) was decreased as compared with that on the other side.

In 1 case with slight motor recovery part of the ulnar area was anesthetic and another part hypesthetic. The two sections showed equal increases in electrical skin resistance (fig. 1A). In another case the ulnar area consisted of a zone of anesthesia and one of hyperesthesia. An increase in electrical skin resistance was present only in the anesthetic area. There were signs of very slight motor recovery (fig. 1B). In a case with definite indications of motor regeneration part of the ulnar area was hypesthetic and another part hyperesthetic. Only the hypesthetic area showed increased electrical skin resistance. One area each of anesthesia, hypesthesia and hyperesthesia was found in a case with definite regeneration. The total anesthetic area and parts of the hypesthetic and hyperesthetic areas showed increased skin resistance (fig. 1C). In a case with advanced motor recovery the total ulnar area was hyperesthetic. Part of this area showed an increase and the remaining part a decrease in skin resistance (fig. 1D).

Thus, anesthetic areas were found in 13 cases. In 7 of these cases there was complete motor paralysis; in 5, beginning motor recovery, and in 1, definite motor regeneration. In 12 cases there was complete correlation of the anesthetic area and the area of increased skin resistance. In 1 case no change in electrical skin resistance was found.

Hypesthetic areas were present in 8 cases. In 4 of these cases slight, and in 4 definite, motor recovery was present. In 5 instances there was a complete correlation of the hypesthetic areas and the area of increased resistance. In 1 case there was a partial correlation, and in 2 no change in the electrical skin resistance of the hypesthetic area was present.

Hyperesthetic areas were found in 4 cases. There were definite motor recovery in 3 cases and slight recovery in 1 case. In 3 cases there was no change in electrical skin resistance in these areas. In 1 case only partial correlation with an area of increased skin resistance was found.

Decrease of electrical skin resistance in the entire affected hand was found in 1 case.

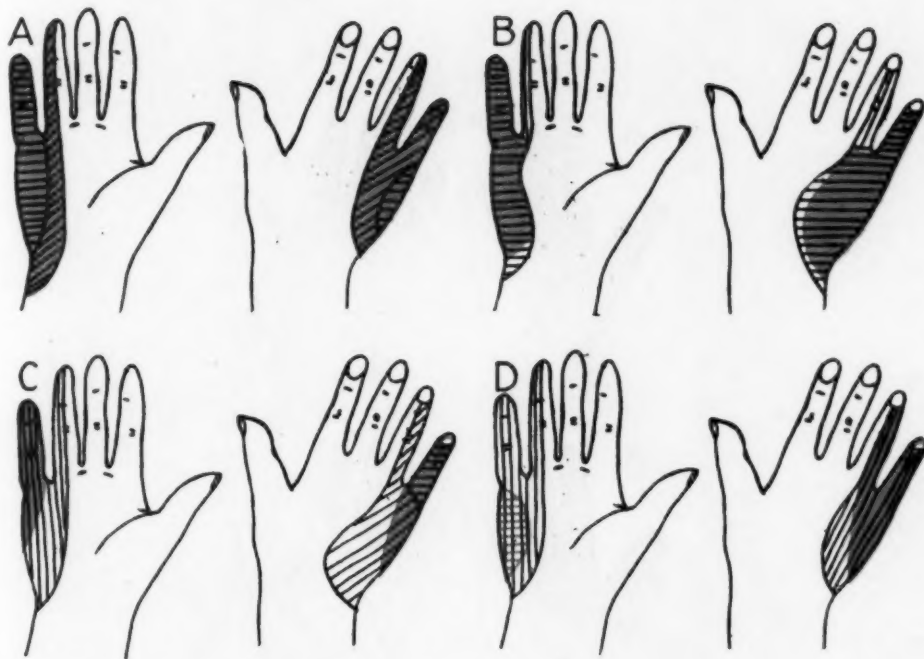


Fig. 1.—*A*, ulnar nerve injury. Part of the ulnar area is anesthetic; another part is hypesthetic. Both zones show increased electrical skin resistance.

B, ulnar nerve injury, with areas of anesthesia and hyperesthesia. The skin resistance is increased only in the anesthetic area.

C, ulnar nerve injury, with zones of anesthesia hypesthesia and hyperesthesia. Increased skin resistance is present in the anesthetic area and in parts of the hypesthetic and hyperesthetic areas.

D, ulnar nerve injury, with hyperesthesia in the whole distribution of the ulnar nerve. Part of this area presents an increase, and another part a decrease of electrical skin resistance.

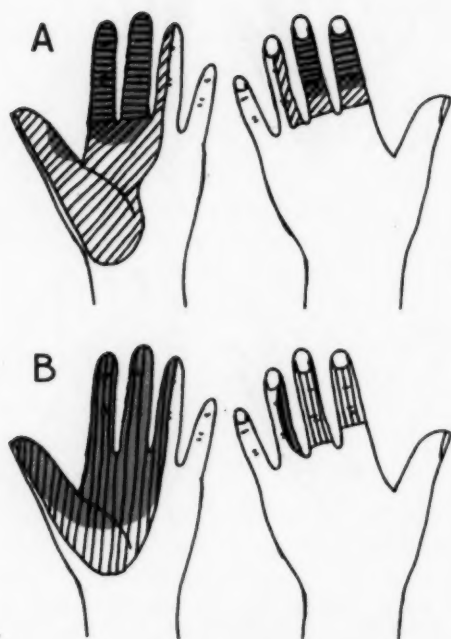


Fig. 2.—*A*, median nerve injury, with anesthetic and hypesthetic areas. In general, the anesthetic area shows increase of electrical skin resistance.

B, median nerve injury, with hyperesthesia in the total area of the nerve. Most of this area shows increase in electrical skin resistance.

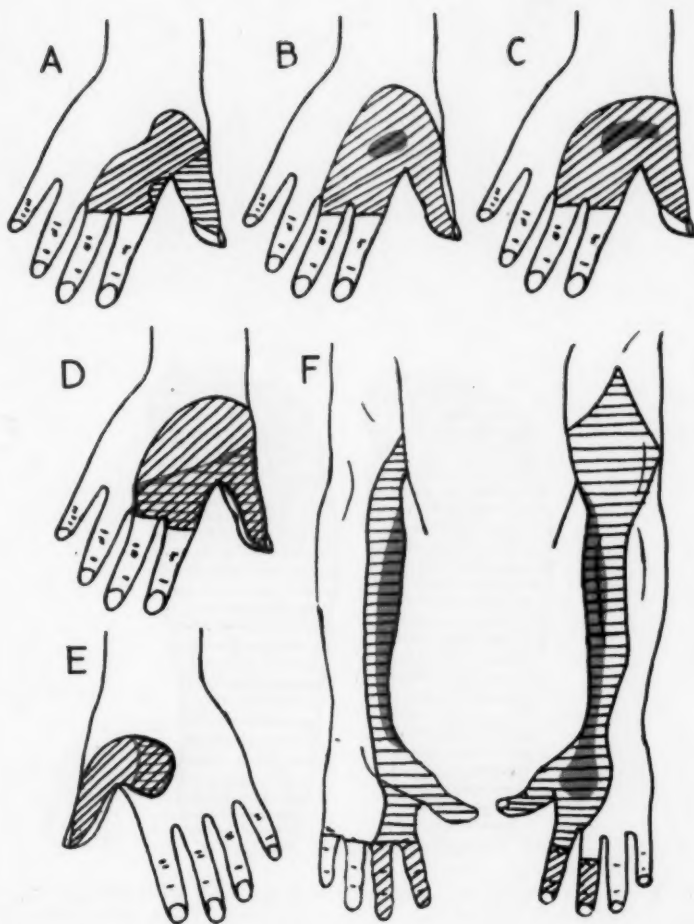


Fig. 3.—*A*, radial nerve injury, with anesthetic and hypesthetic zones. No change in the skin resistance is present.

B and *C*, radial nerve injuries, with hypesthesia and small areas of increased skin resistance.

D, radial nerve injury, with hypesthesia and a partial area of increased skin resistance.

E, radial nerve injury, with partial area of hypesthesia. Only part of this area shows decrease of skin resistance.

F, injury to the brachial plexus with impairment of the median, radial musculocutaneous and posterior cutaneous nerves.

The area of increased skin resistance in the radial zone is larger than that in cases of pure radial nerve injuries.

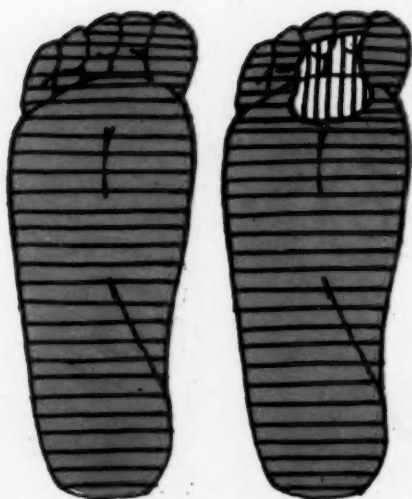


Fig. 4.—Tibial nerve injury. Left, area of anesthesia with correlating increased electrical skin resistance four months after the injury; right, small area of hyperesthesia with normal skin resistance four months after plasma glue suture.

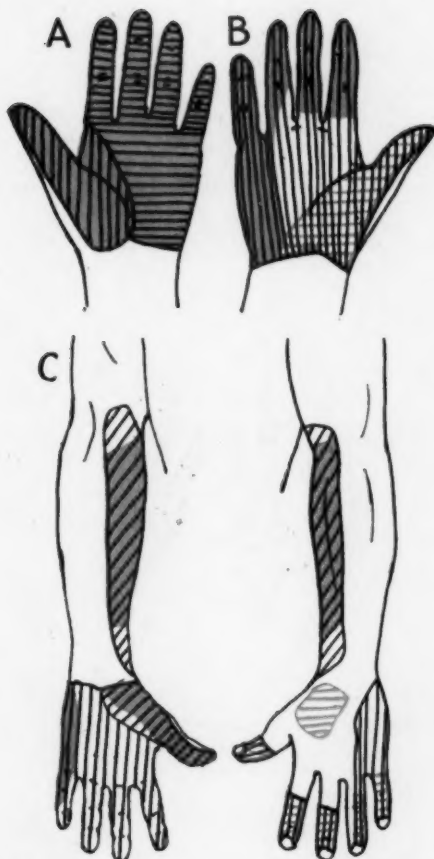


Fig. 5.—*A*, injury to the brachial plexus, with increased skin resistance in the median and ulnar areas. Part of the median area is hyperesthetic, the remaining part and the ulnar area are anesthetic.

B, injury to the brachial plexus with hyperesthesia of the median and ulnar areas. The skin resistance of the ulnar area is increased, the skin resistance of the median area is partly increased, partly decreased and partly normal.

C, injury to the brachial plexus with impairment of the musculocutaneous, median, ulnar and radial nerves. The areas of hypesthesia and hyperesthesia do not correspond to the zones of normal, increased and decreased skin resistance.

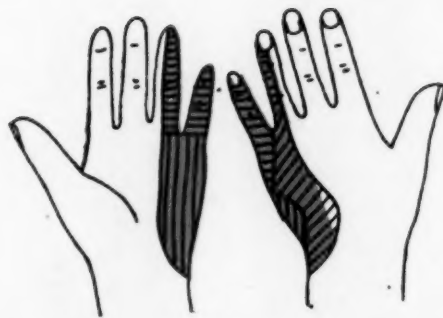


Fig. 6.—Ulnar nerve injury five months after repair of the nerve. Increased skin resistance is present throughout the total ulnar area, with differentiation into anesthetic, hypesthetic and hyperesthetic zones.

Injury to the Median Nerve.—Eighteen cases with injuries of the median nerve were studied. In 6 cases anesthesia of the total area of the nerve was present. In 4 of these cases there was complete motor paralysis with complete neuromuscular degeneration; in 1 case the nerve was in a state of advanced regeneration, and in 1 case no motor impairment was shown. In 5 of these cases there was a complete correlation of the anesthetic area with the area of increased electrical skin resistance. In the case with motor recovery the electrical skin resistance was normal throughout.

In 3 cases areas of both anesthesia and hypesthesia were present in the median zone. In all 3 cases slight or no motor regeneration was shown. All the anesthetic areas showed increased electrical skin resistance. The hypesthetic areas showed increased skin resistance in the whole area in 1 case; there was a partial correlation of the two areas in the second case and no change in skin resistance was found in the third (fig. 2A).

In 4 cases the total median area, and in 1 case only part of the median area, was hypesthetic. In all these cases some degree of motor recovery was present. In 3 cases there was a complete correlation of the hypesthetic area with the area of increased electrical skin resistance. In 1 case only part of the area showed increased electrical skin resistance, and in another no change in skin resistance was present.

In 2 cases part of the median area was hypesthetic and another part hyperesthetic. In both cases progressive motor recovery occurred. In 1 case both zones showed increased electrical skin resistance. In the other case the skin resistance of the entire affected hand was decreased as compared with that of the normal side.

In 2 cases in which the total median area was hyperesthetic there was definite motor recovery. In 1 case there was a decreased skin resistance in the entire affected hand. In the other case most of the hyperesthetic area showed an increased resistance (fig. 2B).

Thus, anesthetic areas were found in 9 cases. In 6 of these cases there was complete motor paralysis; in 2 cases, slight motor recovery, and in 1 case, no motor changes. In 8 cases there was complete correlation of these areas with areas of increased electrical skin resistance. In only 1 case were there no changes in the resistance.

Hypesthetic areas were present in 10 cases. In only 1 of these cases was there complete paralysis; in 4 cases motor recovery was present. In 5 of these cases there was a complete correlation of the hypesthetic areas with the areas of increased electrical skin resistance. In 2 cases there was only a partial correlation. In 3 cases no change in electrical skin resistance was present.

Areas of hyperesthesia occurred in 4 cases, in all of which definite motor recovery was present. In 1 of these cases there was increased

skin resistance in an area correlating with the hyperesthetic area. In another there was only a partial correlation, and in 2 cases the electrical skin resistance was normal.

Decrease of electrical skin resistance in the whole affected hand was found in 3 cases; in 2 of these the entire median sensory area was hyperesthetic, and in the third it was hypesthetic.

Injury to the Radial Nerve.—Fifteen cases with injury to the radial nerve were studied.

An area of anesthesia, and this only partial, was found in 1 case. The remaining part of the radial zone was hypesthetic, and the motor function in this case showed remarkable recovery. No change of electrical skin resistance was found in the radial area as compared with that of the surrounding skin, but the resistance was decreased in the whole affected hand as compared with that on the other side (fig. 3A).

In 13 cases the total radial area was hypesthetic. In 3 of these cases there was complete paralysis; in 3, only slight motor recovery, and in 7, more advanced motor recovery. In only 5 of these cases could a small area of increased skin resistance be found. This was a small, usually half-moon-shaped area, situated slightly above the triangle between the thumb and the forefinger (fig. 3B and C). It was present in 1 case with complete paralysis, in 3 cases with slight motor recovery and 1 case with fairly good motor recovery.

In 7 cases the hypesthetic area did not show any change in electrical skin resistance, and in an additional case a part of the hypesthetic area showed a decrease in skin resistance (fig. 3D).

In 1 case only a part of the radial area presented hypesthesia, and part of this area showed a decreased skin resistance (fig. 3E).

Hyperesthetic areas were not found in the cases of injuries to the radial nerve.

In 4 cases the whole affected hand presented a decrease of skin resistance as compared with that on the other side.

Injury to the Sciatic Nerve.—Sixteen cases with injuries to the sciatic nerve were investigated.

In 11 of these cases there was complete motor paralysis, and in 5 motor recovery was in progress. In 12 cases there was complete anesthesia of the total sensory distribution of the nerve. In 4 cases the anesthetic zone was only partial; in 2 of these 4 cases there was hypesthesia in the remaining zone; in 1 case, hyperesthesia, and in 1 case the tibial area was anesthetic and part of the peroneal area was hyperesthetic. In the last case there were complete paralysis of muscles supplied by the tibial nerve and good motor recovery in the distribution of the peroneal nerve.

There was a complete correlation between areas of increased electrical skin resistance and areas of anesthesia in all cases. In only 1 of the cases with partial hypesthesia was the skin resistance increased in this area. In 2 cases with partial hypesthesia, as well as in the 2 cases with hyperesthetic areas, no increase of skin resistance could be found. In 1 case with a hyperesthetic area there was a correlating area of decreased skin resistance.

Injury to the Peroneal Nerve.—Fourteen cases of injury to the peroneal nerve were investigated.

In 5 of these cases there was complete paralysis; in 2, very slight motor recovery and in 7, advancing motor recovery. In 8 cases the total peroneal area was anesthetic, with complete paralysis in 5 cases and motor recovery in 3 cases. In 7 of these cases there was a complete correlation between the anesthetic areas and the areas of increased skin resistance. In 1 case the area of increased resistance was somewhat smaller than the area of anesthesia.

In 5 cases the whole peroneal area was hypesthetic. In all these cases there was some motor recovery. In 2 cases there was a complete, and in 3 cases only a partial, correlation of the hypesthetic areas and the areas of increased skin resistance. In 1 case with definite motor recovery and a partial area of hypesthesia no change in skin resistance was present. Hyperesthetic areas were not found.

Injury to the Tibial Nerve.—Four cases of injury to the tibial nerve were studied.

In only 1 was there complete paralysis, motor recovery being present to some degree in the others. In all 4 cases the total sensory area was anesthetic and there was a complete correlation of this area with an area of increased electrical skin resistance.

In no case of peripheral nerve injury in the lower extremity was there decrease of skin resistance in the entire affected extremity as compared with that on the other side.

Injury to the Brachial Plexus.—Fifteen cases with injuries to the brachial plexus were studied. Various combinations of complete or partial injuries of the several nerves of the upper extremity were found, but the degree of motor, sensory and autonomic function varied to such an extent that a statistical evaluation seemed impossible. However, certain cases of this series have been used when particular questions could be clarified.

COMMENT

The practical value of the electrical skin resistance test in the evaluation of peripheral nerve injuries depends on the information it gives in addition to the results of examinations of the motor and sensory status. The present investigation was concerned therefore with the

correlation of impairment of the autonomic nervous system, as found with the electrical skin resistance test, with changes in motor and, particularly, sensory function. It has been assumed, predominantly on the basis of the results of sweating tests, that the sensory and the autonomic fibers have a common course in the mixed peripheral nerve and supply approximately the same area of the skin. The question then arises whether the two tests, sensory examination and electrical skin resistance test, yield similar results under the same conditions, whether one test can be substituted for the other or whether the results of the two tests really supplement each other and the variations between the two warrant a careful evaluation of the situation caused by complete or partial nerve injury. In former investigations the degree of sensory loss was somewhat neglected in the correlation of impaired sensory and autonomic function.

In the present evaluation, 87 cases of injuries of the ulnar, median, radial, sciatic, peroneal and tibial nerves were used. Areas of complete sensory loss were found in 51 cases. In 47 of these cases a correlating area of increased skin resistance was shown. This almost complete correlation confirms the findings of Richter and Katz¹ and Jasper and Robb² and demonstrates the value of the electrical skin resistance test in mapping anesthetic areas caused by peripheral nerve injuries.

The evaluation of the results of testing hypesthetic areas is rather complicated. In 40 instances areas of merely partially impaired sensory function were found. In 13 of these cases an area of increased skin resistance coincided with the hypesthetic area. In 11 instances there was only a partial correlation, and in 16 cases no change of skin resistance could be found. This discrepancy between the results of the sensory examination and those of the skin resistance test cannot be neglected. It implies that nerve lesions which are partial, either because the nerve injury was incomplete or because regeneration has taken place, may affect sensory and autonomic functions, respectively, in varying degrees.

The most plausible assumption might be that impairment or recovery of sensory and that of autonomic function have not run parallel. If, for instance, the electrical skin resistance of an area is normal, one might assume that autonomic function has been restored whereas sensory function is still defective. This information would be valuable as an indication of some continuity of the nerve or of some progress in repair. Furthermore, in some cases it was found that the area of increased skin resistance was definitely smaller than the hypesthetic area. Again, it could be stated that partial persistence or restoration of autonomic function existed. These significant conclusions might be justified to some extent when serial tests have been made, but on the occasion of the first examination one should avoid premature conclusions.

Hypesthesia is regarded as evidence of partial impairment of the nerve. But in cases of complete motor paralysis of the radial nerve and known complete section of the nerve, as in cases in which examination was made a short time after plasma glue suture with revision of both nerve ends, only hypesthesia was present in the radial area. The time interval was too short for any sensory regeneration to have taken place, although there may have been "overlap." Of these 8 cases of pure radial nerve injuries a very small area of increased electrical skin resistance was present in only 5. This area might easily have been overlooked (fig. 3 *B* and *C*).

Foerster,¹⁰ after studying the great individual variations in the cutaneous area supplied by the radial nerve, concluded that this nerve does not possess a constant autonomous area. The neighboring areas of the ulnar, median and dorsal and lateral antibrachial cutaneous nerves apparently "overlap" the area of the radial nerve to a varying degree. The small half-moon-shaped area of increased resistance which we found coincides with the area of anesthesia which Foerster found in some of his cases. The area of increased resistance in the zone of the radial nerve may become larger when the neighboring nerves are involved. In a case of injury to the brachial plexus (fig. 3 *F*) a larger area of increased electrical skin resistance was demonstrated in an extensive area of sensory loss due to involvement of the median, radial, posterior cutaneous and musculocutaneous nerves.

It would seem, therefore, that there is sufficient evidence to indicate not only that the sensory area of the radial nerve is variable and lesions of this nerve cause mainly a hypesthesia, but also that the autonomic fibers are few and lesions of this nerve alone do not produce areas of increased skin resistance in a large proportion of cases. The absence of changes in electrical skin resistance in cases of injuries to the radial nerve is thus of no significance.

In instances of injuries of other nerves hypesthesia may also not be due to partial impairment or to regeneration of the nerve which supplied this area with sensory fibers. The affected nerve may be completely injured but part of the sensory area may be functioning to some extent. Foerster¹⁰ differentiated between the "autonomous areas," exclusively supplied by one nerve, and the "maximal area," which remains when all neighboring nerves to this region are severed. The term "mixed" or "intermediate area" is applied to the zone of multiple sensory innervation. The "subsidiary area" of a nerve is that region of its sensory innervation aside from its autonomous area (i. e., the extension into the "mixed area"). The difference between

10. Foerster, O., in Lewandowsky, M.: *Handbuch der Neurologie*, Berlin, Julius Springer, 1929, supp., pt. 2.

autonomous areas and maximal area, or the average extension of sensory defects, as elaborated from various multiple nerve lesions, is shown in figure 7 (from Foerster¹⁰). Pollock¹¹ and Weddell, Guttmann and Guttmann¹² pointed out that soon after nerve section the area of sensory loss undergoes progressive shrinkage. This shrinkage was regarded as due to "overlap" and concerns chiefly the fibers for pain sense. This situation was further studied by Guttmann and Highet,¹³ who found that this "progressive shrinkage of the area of sensory loss by nerve overlap [occurs] at a time when there was no possibility of true recovery by regeneration." It was also noted by Guttmann¹⁴

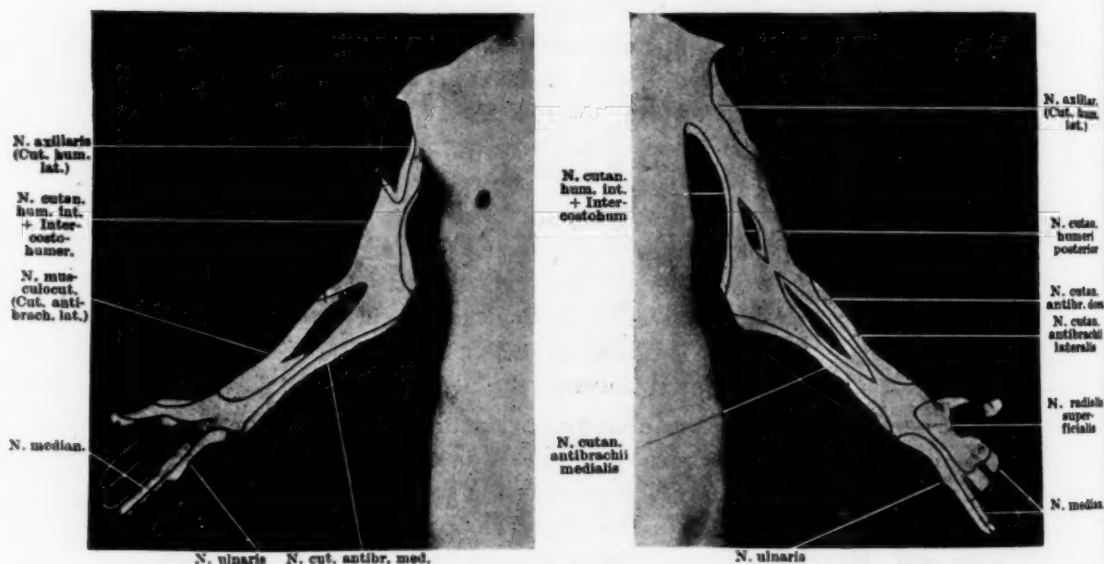


Figure 7

that the overlaps of pain sensation and sweating function are "conspicuous and similar." Guttmann and Guttmann¹⁵ finally concluded,

11. Pollock, L. J.: Nerve Overlap as Related to the Relatively Early Return of Pain Sense Following Injury to the Peripheral Nerves, *J. Comp. Neurol.* **32**:357 (Dec.) 1920.

12. Weddell, G.; Guttmann, L., and Guttmann, E.: The Local Extension of Nerve Fibers into Denervated Areas of Skin, *J. Neurol. & Psychiat.* **4**:206 (July-Oct.) 1941.

13. Guttmann, L., and Highet, W. B., cited by Highet.¹⁶

14. Guttmann, L.: Topographic Studies of Disturbances of Sweat Secretion After Complete Lesions of Peripheral Nerves, *J. Neurol. & Psychiat.* **3**:197 (July) 1940.

15. Guttmann, E., and Guttmann, L.: Factors Affecting Recovery of Sensory Function After Nerve Lesions, *J. Neurol. & Psychiat.* **5**:117 (July-Oct.) 1942.

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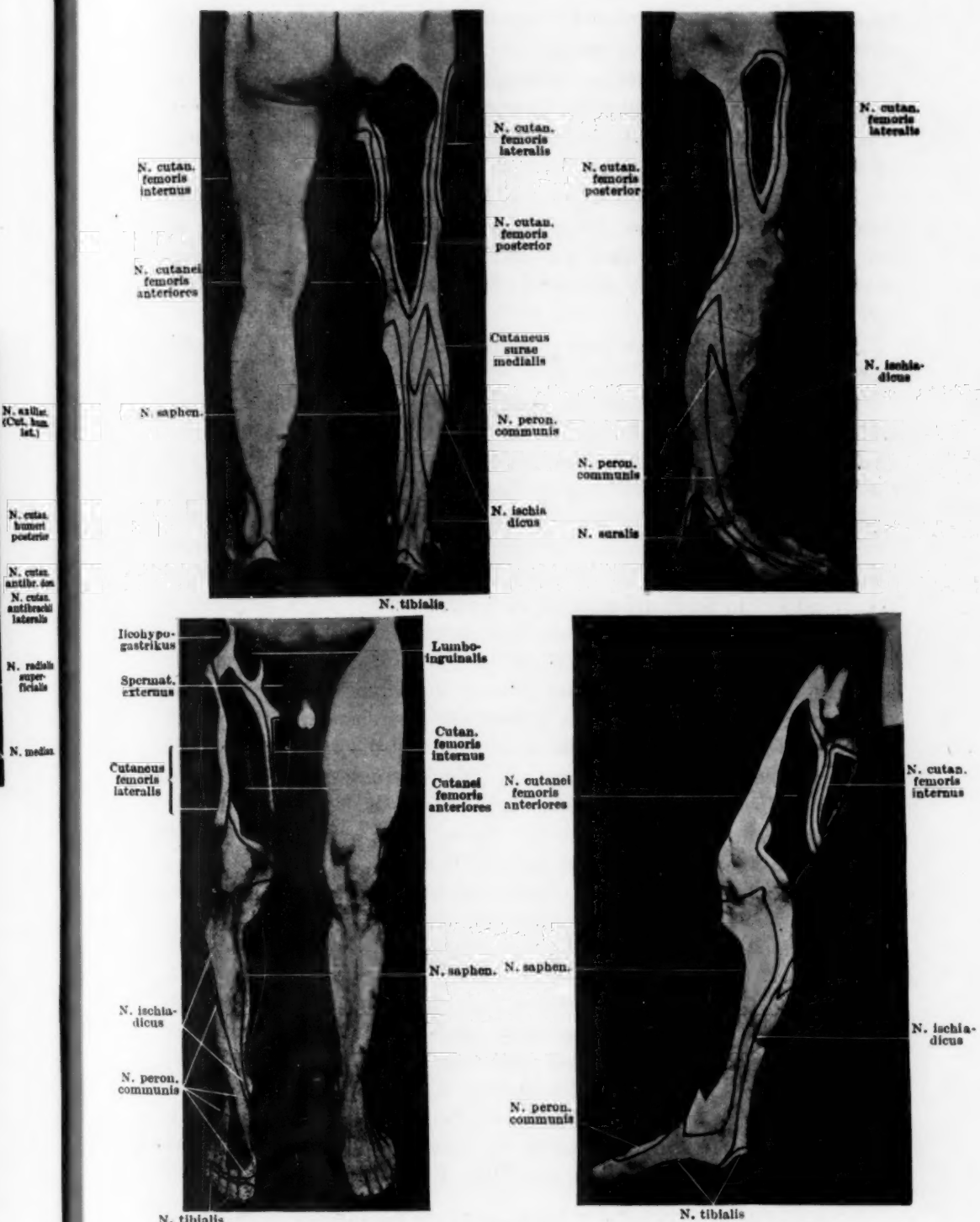


Fig. 7.—“Autonomous” and “maximal” sensory areas (after Foerster¹⁰).

by means of animal experiments, that the complex process of recovery of sensation has three components:

1. "Recovery" in zones of "overlap," or intermediate zones, by the resumption of "readjustment" of function by fibers of adjacent nerves. The nature of this process is at present obscure and is fully discussed by these authors.

2. Anatomic extension of fibers from adjacent nerves into the denervated area, even into the autonomous zone. This extension of neighboring nerve fibers into the area of the interrupted nerve was demonstrated histologically in animal experiments by Wedell, Guttmann and Gutmann.¹²

3. Regeneration of the interrupted nerve, or "true" sensory regeneration.

Highet,¹⁶ using a technic of peripheral nerve block with procaine, and especially blocking the neighboring nerves of the nerve in question, studied the difference in size of the maximal and autonomous zones and the problems of anomalous sensory loss and "recovery" by means of "overlap."

The aforementioned findings are extremely important in the evaluation of hypesthetic areas and of areas without increase in the electrical skin resistance in the distribution of an injured nerve. Thus, restoration of sensory and autonomic function may be due to subsidiary function (physiologic or anatomic overlap) of neighboring nerves, particularly when it is found in intermediate areas and encroaching only on the autonomous area. Furthermore, it is quite possible that sensory and autonomic functions may be reinnervated in this manner in a variable or nonparallel degree. Consequently, the reappearance of normal electrical skin resistance need not be always or solely due to true regeneration of the autonomic nerve fibers. The case in figure 4 is an example of the possibility of subsidiary autonomic function of the neighboring nerve. The tibial nerve was injured on Feb. 23, 1945. Figure 4, left, shows the area of anesthesia and increased electrical skin resistance found on June 15. In July 1945 a plasma glue suture was performed. On Nov. 6, 1945 the patient was reexamined, and a small area of hyperesthesia was found on the plantar surfaces of the second and third toes and the interspace between these toes; skin resistance was normal in a correlating area (fig. 4, right) which is probably a subsidiary area of the distribution of the deep peroneal nerve. In these instances only investigation of the neighboring nerves with the nerve block technic could clarify the situation and demonstrate the true innervation of the area in question. Furthermore, the extent of

16. Highet, W. B.: Procaine Nerve Block in the Investigation of Peripheral Nerve Injuries, *J. Neurol. & Psychiat.* 5:101 (July-Oct.) 1942.

the autonomous areas of the autonomic distribution of the peripheral nerves has still to be determined more definitely. Thus, one can conclude that the presence of an area of normal skin resistance in the distribution of an injured nerve is not a definite indication of a partial lesion or a sign of regeneration.

In 10 cases hyperesthetic areas were found. In 5 cases the electrical skin resistance was normal. In 2 cases the hyperesthetic area coincided with the area of increased skin resistance, and in 1 case there was only a partial correlation. In 1 case the hyperesthetic area showed a decrease of skin resistance. In 1 case in which the total ulnar area was hyperesthetic zones of both increased and decreased skin resistance were present in this area. In addition, in 2 of these 10 cases the whole affected hand showed a decreased skin resistance.

In some cases of injury of the brachial plexus with hyperesthetic zones the correlation of the sensory status and the skin resistance was complicated. Three cases will illustrate this problem. In case 1 (fig. 5 *A*) the skin resistance was increased in the median and ulnar distribution, but part of the median area was hyperesthetic and the remaining part and the ulnar area were anesthetic. In case 2 (fig. 5 *B*) there was hyperesthesia of the median and ulnar areas of the entire palm; the skin resistance of the ulnar area was increased, and that of the median area was partly increased, partly decreased and partly normal. In case 3 (fig. 5 *C*), an instance of injury to the brachial plexus with impairment of the musculocutaneous, median, ulnar and radial nerves, the areas of hypesthesia and hyperesthesia did not correspond to the areas of normal, increased and decreased skin resistance.

From our findings it can be concluded that there is no definite relation between changes in electrical skin resistance and areas of hyperesthesia.

A decrease in skin resistance of the affected area as compared with that of the surrounding areas of the same extremity was found in only 4 cases, in all of which definite motor recovery took place. In 2 of these cases the sensory areas were hyperesthetic, and in the other 2, hypesthetic. A decrease in skin resistance of the entire affected hand occurred in 8 cases (4 of radial, 3 of median and 1 of ulnar nerve injury). In all these cases there was evidence of motor and sensory recovery. In 6 instances there was hypesthesia, in 1 case hyperesthesia, and in 1 case both hyperesthetic and hypesthetic areas were present.

Thus, there are two types of circumstances in which decrease in skin resistance occurs; different underlying mechanisms may be responsible. It is quite possible that during the course of regeneration an "irritative" process causes autonomic hyperfunction in the area of distribution of the involved nerve. When the entire hand is involved, a

more complex "reflex" mechanism may play a role (Guttmann¹⁴), but the nature of such a mechanism is quite uncertain.

An attempt was also made to correlate autonomic with motor function. In the series of 87 cases there were 36 with complete neuromuscular degeneration. Of the 47 cases with anesthetic zones and complete correlation with skin resistance, neuromuscular degeneration was complete in only 29, there being signs of motor recovery in the other 18 cases. This indicates that motor recovery, as evaluated by chronaxia, may be an earlier and more sensitive indicator of nerve regeneration. In 51 cases there was evidence of motor recovery and no real correlation with sensory or autonomic function. It should be mentioned that anomalous innervation of muscles by neighboring nerves or supplementary and "trick" movements and local extension of nerve fibers (from neighboring nerves) into denervated muscle (Van Harreveld¹⁷) must be considered when motor recovery is being evaluated. The nerve block technic of Highet¹⁸ may also be used to solve these problems.

Course of Regeneration.—The entire course of regeneration, from the time of injury to the complete restoration of function, could not be evaluated in this series, as insufficient time has elapsed. However, the investigation of intermediate stages revealed certain principles which should be valuable in further studies. It is evident that all three functions—motor, sensory and autonomic—should be considered significant, as it has been shown that in many cases there is no correlation in the degree of recovery of these functions. Generally, recovery of motor function seems to be the earliest sign of regeneration, particularly in cases of injury to the nerves of the lower extremity, but also to some extent in cases of high lesions of nerves of the upper extremity in which the distance between the lesion and the muscles to be reinnervated is shorter than that between the lesion and the sensoriautonomic areas. Also, as previously discussed, the restoration of sensory and autonomic function in the area of the distribution of a certain nerve may not necessarily indicate regeneration of sensory and autonomic fibers of the injured nerve.

Increased electrical skin resistance was in all cases an indication of injured nerve function. The lack of increased skin resistance in the distribution of a definitely injured nerve does not necessarily mean the appearance of regeneration; it was noted especially with total injuries of the radial nerve and with partial lesions of other nerves.

In some cases the sensory examination revealed definite signs of recovery, whereas the skin resistance remained as high as before.

17. Van Harreveld, A.: Re-Innervation of Denervated Muscle Fibers by Adjacent Functioning Motor Units, *Am. J. Physiol.* **144**:477 (Sept.) 1945.

In a case of complete ulnar nerve injury at the wrist examination, five months after repair, showed that skin resistance was increased to the same degree throughout the entire ulnar area but that there were zones of hypesthesia and hyperesthesia, in addition to a small area of anesthesia (fig. 6).

The appearance of a decrease of skin resistance in the area of an injured nerve might be an indication of definite regeneration, but enough data of this type have not yet been collected to warrant definite conclusions. Thus, examination of skin resistance alone does not always give the entire available information concerning the degree of regeneration at a given time. Serial qualitative and, especially, quantitative studies of the degree of increase and diminution of this value during recovery will lend a higher accuracy to the use of the electrical skin resistance test.

Foerster¹⁰ has pointed out that the restitution of sensory function requires a much longer time than motor function. There is nothing known as yet about the regeneration of autonomic fibers except for the work of Neumann, Grundfest, Berry, Rule and Cohn,¹⁸ who showed that after the section and suture of the sciatic nerve of the cat fifteen to thirty weeks elapsed before the first signs of sweat production were observed. The complicated situation with partial nerve injuries and during regeneration indicates that one should not rely too much on the results of the skin resistance test until more is known about the rate and mode of autonomic regeneration.

SUMMARY

The electrical skin resistance test was evaluated in 87 cases of injuries of the median, ulnar, radial, sciatic, peroneal and tibial nerves. An additional 15 cases of injuries of the brachial plexus were studied. The results of the skin resistance test were correlated with the degree and area of sensory impairment and the degree of motor dysfunction.

In cases of complete nerve injury the area of increased electrical skin resistance coincided well with the area of sensory loss and the degree of motor paralysis. The skin resistance test is therefore valuable in mapping anesthetic areas.

In cases of partial nerve lesions, due either to incomplete injury or to some degree of recovery, there was no regular correlation of the electrical skin resistance, the hypesthetic or hyperesthetic areas and the motor status. Fundamental physiologic and anatomic mechanisms responsible for this irregularity are discussed.

18. Neumann, C.; Grundfest, H.; Berry, C. M.; Rule, C., and Cohn, A. E.: Return of Function of Sweat Glands After Cutting or Crushing Sympathetic Nerves, *Proc. Soc. Exper. Biol. & Med.* **54**:27 (Oct.) 1943.

The appearance of decreased electrical skin resistance in the area of distribution of the injured nerve and the whole affected hand is discussed.

Certain principles in the evaluation of the course of regeneration with the skin resistance method are considered.

The electrical skin resistance test does not give definite information for the diagnosis and evaluation of partial dysfunction of peripheral nerves. In our material examination of motor and sensory functions revealed more practical data. However, the continued study of the electrical skin resistance can certainly advance knowledge of autonomic innervation.

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PRIMARY BEHAVIOR DISORDERS AND PSYCHOPATHIC PERSONALITY

I. Correlations of the Electroencephalogram with Family History and Antecedent Illness or Injury

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IN PREVIOUS publications concerning the electroencephalographic evaluation of primary behavior disorders in children¹ and of psychopathic personality in adults,² we reported correlations with age, sex, family history and antecedent severe illness or cerebral injury of early childhood, which previously had not been considered of etiologic significance. Statistical analyses revealed that electroencephalographic abnormality was unrelated to age and sex but was related to either a "positive" family history or a personal history of cerebral trauma or severe illness. The inference was that the abnormal electroencephalogram was of either genogenic or histogenic origin and thus represented some aspect of the neural limits of the organism. The original number of patients under study has been increased, thus permitting more complete statistical analyses, from which further implications may be drawn. The present communication has this for its purpose.

Because of the consistency of the findings in the two previous studies and the clinical similarities of the two diagnostic groups, study of the groups singly and in combination would seem warranted. The patients designated as having primary behavior disorders were characterized, in the main, not by a single simple habit or disturbance of conduct but by a multiplicity of these disturbances. Only the patients with the more serious behavioral problems were brought to the hospital, as those with

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1. Gottlieb, J. S.; Knott, J. R., and Ashby, M. C.: Electroencephalographic Evaluation of Primary Behavior Disorders in Children: Correlations with Age, Sex, Family History and Antecedent Illness or Injury, *Arch. Neurol. & Psychiat.* **53**:138-143 (Feb.) 1945.

2. Knott, J. R., and Gottlieb, J. S.: Electroencephalographic Evaluation of Psychopathic Personality: Correlations with Age, Sex, Family History and Antecedent Illness or Injury, *Arch. Neurol. & Psychiat.* **52**:515-519 (Dec.) 1944.

the less severe disorders had been cared for by various therapeutic agencies. As a result, then, the groups of patients designated as having either primary behavior disorders or psychopathic personality were characterized on the behavioral level by outspoken social maladjustment, which had been either continuous or repeatedly recurrent over a relatively long period. The chief symptoms were one or more of the following: delinquency and law breaking, socially unconventional behavior, emotional instability or other affective liabilities and/or aberrant sexual behavior. There were stereotyped deviations in the moral, social, sexual and/or emotional components of their personalities. The maladjustment in the patients of both groups, furthermore, was not attributable to defects in intelligence, structural diseases of the brain, epilepsy, psychoneuroses or psychoses.

These two diagnostic groups were separated arbitrarily on the basis of age, disturbances in patients of 16 years or above being considered as psychopathic personality and disturbances in patients of 15 years or below as primary behavior disorders. Thus, on a descriptive level, the concept of psychopathic personality became an extension of the concept of primary behavior disorders in children with reference to the age factor—this, irrespective of the recognition that the prognosis for some members of the younger group was better than the prognosis for members of the older group—a difference which may be related to therapeutic intercession nearer the origin of the behavioral difficulty; to the plasticity of the developing personality of a younger person, in contrast to the rigidity of the personality of the older one, and/or to the differences in etiologic composition of the constellations of factors leading to the development of these categories.

The definitions are so broad as to be indicative of a heterogenous collection of disturbances in behavior and cannot be considered adequate for clinical entities. Both primary behavior disorders and psychopathic personality may be considered categories composed of a number of conditions. The composition of each category in terms of the types of clinical conditions and their relative proportions may, and probably do, vary. By comparative studies, differences may be obtained and evaluated, with the ultimate expectation of better definition, etiologic, as well as descriptive; of better understanding of the composition of the categories, and, possibly, of segregation of clinical entities.

METHOD

The present method of study has been described in considerable detail elsewhere.³ Here it has been applied to 100 patients with primary behavior disorders and 100 patients with psychopathic personality for whom detailed psychiatric, physical, neurologic, laboratory, psychometric and social records had been

3. Gottlieb, Knott and Ashby.¹ Knott and Gottlieb.²

obtained. These patient populations were maintained as homogeneous as possible by the exclusion of all with questionable diagnoses. No patient was included whose symptoms were suspected of being related to epilepsy, psychoneurosis, psychosis or sequelae of a physical illness or injury or whose intelligence quotient was below 80.

Certain data were selected and used as criteria for the designation of the family history as positive. There were four ancestral types which thus could be clearly defined and labeled on the basis of the presence of one of the following conditions: (1) psychosis, (2) maladjusted personality, (3) chronic alcoholism and (4) epilepsy. The use of the term "psychosis" needs no explanation except to say that in many instances the type of such disorder could not adequately be determined through the use of the historical method. The term "maladjusted personality" was applied to those relatives for whom there was evidence of severe social maladjustment (nonpsychotic) and in whom there was apparent difficulty in emotional control. Although the term "psychopathic personality" could not be used, as few of the antecedents had had clinical examinations, the descriptions of their personalities and the histories of their apparent chronic inadequate adaptation to society resembled this diagnostic category. Chronic alcoholism was almost always only one aspect of a severe personality disturbance. In fact, the personalities and the types of adjustment of persons with chronic alcoholism resembled those of the patient designated as "maladjusted personality." Only the chronic addiction to alcohol allowed their designation in a separate category. The term "epilepsy" needs no explanation.

The antecedent illnesses and injuries which were selected were: (1) prematurity; (2) birth injury or questionable birth injury (actually no definite instance was included; the cases placed in this category were all in question, the historical evidence indicating mainly prolonged labor or difficulty in delivery); (3) head injury complicated by unconsciousness; (4) severe illness complicated by delirium, coma or severe stupor (on the basis of the historical material, these illnesses could be divided into very severe and moderately severe, the term "very severe" referring to those illnesses complicated by delirium or coma and meningeal irritation and followed by regression in development and the term "moderately severe" referring to those illnesses complicated by only delirium or coma); (5) convulsions in infancy not considered epileptic in origin, and (6) a period of anoxemia, either at birth or later in life.

Six lead electroencephalograms were obtained with a Grass ink-writing oscillograph in the usual way from virtually all the patients (for the small minority three lead records were made). The records of the patients 15 years of age or under were classified according to the method described in a previous publication.⁴ Essentially, the records were classified as normal or other than normal, depending on their similarity to age frequency limits of neurologically screened normal children, as determined by Lindsley⁴ and Gibbs and Gibbs.⁵ If the frequencies were within these defined limits, the record was considered as normal. If in addition there were some nondominant too slow or too fast frequencies, the record was considered as questionably normal. (Such records were not included in the final statistical treatment of these data because of their uncertain allocation.) If the

4. Lindsley, D. B.: A Longitudinal Study of the Occipital Alpha Rhythm in Normal Children: Frequency and Amplitude Standards, *J. Genet. Psychol.* **55**: 197-213 (Sept.) 1939.

5. Gibbs, F. A., and Gibbs, E. L.: *An Atlas of Electroencephalography*, Cambridge, Mass., Lew A. Cummings Co., 1941.

dominant frequencies were above or below the age norm recurrently, continuously or paroxysmally, the record was classified as abnormal. The records of the patients 16 years of age or over were classified according to the frequency scale of Gibbs, Gibbs and Lennox.⁶ A 97 per cent agreement was obtained by one of us (J. R. K.) with the ratings of Dr. F. A. Gibbs on the rereading of records which he loaned to us.

All records were obtained from the patients in the waking state, with closed eyes but without hyperventilation.

DATA

The electroencephalograms for both the 100 patients with primary behavior disorders and the 100 patients with psychopathic personality are presented in table 1. All electroencephalographic patterns not

TABLE 1.—Incidence of Electroencephalographic Patterns with Relation to Primary Behavior Disorders and Psychopathic Personality

Category *	Number or per Cent	Total Incidence of Abnormal Patterns
Primary Behavior Disorders		
N.....	37	56
QN.....	7	
RA.....	31	
CA.....	11	
P.....	14	
Total.....	100	
Psychopathic Personality		
N.....	42	58
S ₁	43	
F ₁	3	
S ₂	9	
F ₂	1	
P.....	2	
Total.....	100	

* In accordance with both the electroencephalographic classification for children described under "method" and the classification for adults of Gibbs, Gibbs and Lennox,⁶ N indicates a normal; QN, a questionably normal; RA, a recurrently abnormal; CA, a continuously abnormal, and P, a paroxysmal electroencephalographic pattern. S₁ indicates moderate amount of activity slower than 8½ cycles per second in any lead; F₁, moderate amount of activity faster than 12 cycles per second in any lead; S₂, great amount of activity slower than 8½ cycles per second in any lead, and F₂, great amount of activity faster than 12 cycles per second in any lead.

considered as normal or questionably normal were designated as abnormal and for statistical purposes were treated as one category. Although the classificatory scales differ for the two diagnostic groups and the results can be compared directly only in part, the percentages of abnormality, 56 and 58, respectively, were very similar. These percentages are considerably higher than those reported for presumably normal children and adults. For a group of 270 unscreened children on whom records were made by one of us (J. R. K.) the percentage of abnormality was approximately one-quarter the value of that for

6. Gibbs, F. A.; Gibbs, E. L., and Lennox, W. G.: Electroencephalographic Classification of Epileptic Patients and Control Subjects, *Arch. Neurol. & Psychiat.* 50:111-128 (Aug.) 1943.

the group of problem children. The most comprehensive electroencephalographic study of normal children has been made by Henry.⁷ Through a personal communication, he indicated that his percentage of abnormal records was approximately one half of ours when essentially the same classification was used. Since the classificatory scale used for adults is the one described by Gibbs, Gibbs and Lennox,⁸ comparisons may be made with the data obtained by these authors on neurologically screened subjects. For 1,000 of these subjects they reported an incidence of 15.8 per cent abnormal electroencephalograms. This is approximately one-fourth the occurrence of abnormality in our material.

One difference in the electroencephalographic abnormalities between patients with primary behavior disorders and patients with psychopathic personality is presented in table 1. There were 14 patients with primary behavior disorders who had paroxysmally abnormal electroencephalograms, whereas only 2 of the patients with psychopathic personality had similar electrocortical potentials. This difference has considerable meaning in terms of probabilities. Whereas a control normal adult population has an incidence of 0.9 per cent of paroxysmal waves, an epileptic population has at least 30 per cent.⁹ Thus, a paroxysmally abnormal electroencephalogram would seem to indicate a high relationship to epilepsy. The inference seems clear that in spite of an attempt to eliminate all patients with epilepsy the younger group may contain patients who do not as yet show specific symptoms but who are potentially epileptic. The adult group may contain fewer such patients, since the aging process would assist in selectively preventing patients from being included in this group.

Although the distribution of the electroencephalographic patterns for patients with primary behavior disorders may suggest the inclusion of a small number who are potentially epileptic, the majority cannot be thought of as belonging to that clinical category. The high incidence of abnormal electroencephalograms for patients in the two diagnostic groups suggests that the cause of this abnormality must be sought elsewhere. So far as is known, permanent or relatively long-lasting electroencephalographic activity outside normal limits has been ascribed only to factors of inheritance, injury or infection of the brain and disturbances of cerebral metabolism. It, therefore, becomes pertinent to examine the historical material in terms of a positive family history and antecedent severe illness or cerebral injury to ascertain (1) the incidence of these factors with relation to the two conditions and (2) their relation to the abnormal electroencephalogram.

7. Henry, C. E.: *Electroencephalograms of Normal Children*, Monographs of the Society for Research in Child Development, Washington, D. C., National Research Council, 1944, vol. 9, no. 3, 1-71.

First, the incidence of the components both of a positive family history and of a personal history of severe illness or cerebral injury may be considered. The data are presented in table 2. The components, or factors, for a positive family history are epilepsy, maladjusted personality, alcoholism and psychosis. In the analysis, both direct and collateral lines were included. The components, or factors, for illness or injury are anoxia, convulsions, head injury, severe illness, birth injury and prematurity. Irrespective of the number of times any one factor occurred in a patient's history, it was recorded but once. When two or more factors occurred, they were so recorded. Thus, there was a total of 80 factors in the family histories for 50 patients with

TABLE 2.—Incidence of the Components or Factors of Positive Family History, Antecedent Severe Illness and Cerebral Injury Among Patients with Primary Behavior Disorders or Psychopathic Personality

	Primary Behavior Disorders		Psychopathic Personality		Totals	
	No. of Patients	No. of Factors	No. of Patients	No. of Factors	No. of Patients	No. of Factors
Family history						
Epilepsy.....		8		5	13 (6.5%)	
Maladjusted personality...		33		37	70 (35.0%)	
Alcoholism.....		19		21	40 (20.0%)	
Psychosis.....		20		23	43 (21.5%)	
Totals.....	50	80	59	86	109 (54.5%)	
Illness or injury						
Anoxia.....		0		2	2 (1.0%)	
Convulsions.....		10		4	14 (7.0%)	
Head injury.....		10		10	20 (10.0%)	
Severe illness.....		38		15	53 (26.5%)	
Birth injury.....		19		5	24 (12.0%)	
Prematurity.....		5		2	7 (3.5%)	
Totals.....	55	82	31	38	86 (43.0%)	

primary behavior disorders and a total of 86 factors for 59 patients with psychopathic personality. In other words, some of the family histories, as well as some of the personal histories of illness or injury, contained two or more factors. There were 8 patients with primary behavior disorders and 5 patients with psychopathic personality who had epileptic relatives. When the two diagnostic groups were combined the percentage of patients with epileptic relatives was 6.5. The incidences of maladjusted personality, alcoholism and psychosis in the family histories were remarkably similar for the two diagnostic groups (33, 19 and 20 and 37, 21 and 23 per cent respectively). Thus, the incidence of a family history of maladjusted personality was about twice that of alcoholism and psychosis and about four times that of epilepsy.

The higher incidence of factors of illness and injury in the group with primary behavior disorders is quite evident on examination of the lower half of the table. There was a total of 82 factors of illness or injury for 55 patients with primary behavior disorders, in contrast

to a total of 38 factors for 31 patients with psychopathic personality. The incidences of severe illness, questionable birth injury and convulsions were considerably greater for the younger than for the older population (38, 19 and 10 and 15, 5 and 4 per cent, respectively). The incidences of head injury were the same for the two groups (10 per cent each). This greater incidence of illness and injury for the patients with primary behavior disorders suggests that there may be included within this group a larger number of patients whose adjustment is related to these factors⁸ than is to be found among the psychopathic personalities.

TABLE 3.—*Analysis of the Electroencephalogram with Relation to Antecedent Severe Illness and/or Head Injury and Positive Family History*

	Electroencephalograms							
	Abnormal		Question-ably Normal		Normal		Total Incidence	
	No.	%	No.	%	No.	%	No.	%
	Primary Behavior Disorders							
Positive family history only.....	14	56	5	20	6	24	25	25
Previous illness and/or injury only.....	19	63	11	37	30	30
Positive family history and previous illness and/or injury	15	60	1	4	9	36	25	25
No positive family history or previous illness or injury.....	8	40	1	5	11	55	20	20
Totals.....	56	56	7	7	37	37	100	100
Psychopathic Personality								
Positive family history only.....	29	65	16	35	45	45
Previous illness and/or injury.....	10	59	7	41	17	17
Positive family history and previous illness and/or injury	11	79	3	22	14	14
No positive family history or previous illness or injury.....	8	33	16	67	24	24
Totals.....	58	58	42	42	100	100
Both Conditions								
Positive family history only.....	43	62	5	7	22	31	70	35
Previous illness and/or injury only.....	29	62	18	38	47	23.5
Positive family history and previous illness and/or injury	26	67	1	2	13	31	39	18.5
No positive family history or previous illness or injury.....	16	36.5	1	2.5	27	62	44	22
Totals.....	114	57	7	3.5	79	39.5	200	100

Second, an analysis of the relation of the electroencephalographic patterns to the categories of positive family history and previous severe illness or cerebral injury in the two groups of patients may now be considered. The data are presented in table 3. The first part presents the data for patients with primary behavior disorders; the second, for

8. The difference in the incidence of illness and injury may be related to the historical method: The younger the patient, the more likely is a severe illness or injury to be recalled by relatives and reported by them. Because of this tendency, further critical comparisons are indicated, which will be discussed in a subsequent publication.

patients with psychopathic personality, and the third for a combination of the two. The incidence of the factors of positive family history and previous illness and injury was classified under four categories: (1) positive family history only, (2) previous severe illness and/or cerebral injury only, (3) positive family history and previous severe illness and/or cerebral injury, and (4) absence of positive family history or previous severe illness or cerebral injury. The incidence of these categories for the two groups of patients may be compared. When the categories of positive family history only and positive family history and previous illness and/or injury were combined for the two groups of patients, the total incidence of positive family history became 50 per cent for primary behavior disorders and 59 per cent for psychopathic personality. A similar comparison may be made for the incidence of previous illness and/or injury. When the two categories of previous illness and/or injury only and positive family history and previous illness and/or injury were combined, the incidence of previous illness and/or injury was 55 per cent for primary behavior disorders and 31 per cent for psychopathic personality. Again, this indicates the greater incidence of illness and/or injury in the patients of the former group.

The relation of the electroencephalogram to the categories for the two groups of patients, single and combined, may now be considered.

In the group of primary behavior disorders, 14 children with positive family history only had abnormal electroencephalograms, and 6 had normal records. Eight children with negative family and personal histories had abnormal electroencephalograms, and 11 had normal records. By subjecting these two distributions to the chi square test (χ^2)⁹ for independence, it was found that a level of significance of 5 to 10 per cent was obtained. This means that the two distributions would be expected to differ from 5 to 10 times in 100 series of observations. Hence, this difference may be regarded as marginally significant.

Nineteen children giving a positive personal history of illness and/or injury showed abnormal electroencephalograms, and 11 had normal records. When this distribution is compared with that for the group with negative family and personal histories, a level of significance of 10 to 20 per cent is obtained. This is not sufficiently great to suggest that the two groups differ significantly.

When the 15 children with abnormal electroencephalograms who presented both a positive family history and a personal history of illness and/or injury and the 9 children with normal records but with similar histories were compared with the group with negative family and personal histories the level of significance obtained was

9. Lindquist, E. F.: *Statistical Analysis in Educational Research*, New York, Houghton Mifflin Company, 1940, p. 41.

from 10 to 20 per cent. These two groups, therefore, did not differ more than may be accountable by chance.

In contrast, for the patients with psychopathic personality, the distributions of all three of the categories with positive factors were significantly different from the distribution of the category without positive factors. The levels of significance, as revealed by the χ^2 test, were between 1 and 2 per cent.

When the group of patients with psychopathic personality and that with primary behavior disorders were combined, the distributions of all three of the categories with positive factors were significantly different from the distribution of the category without positive factors. The levels of significance, as revealed by the χ^2 test, were then between 1 and 2 per cent. This combination of groups appeared justified in view of the fact that a statistical comparison of each subgroup (positive family history, positive personal history, combined positive family and personal histories, and negative history) for the patients with primary behavior disorders and psychopathic personality yielded levels of significance which made it unnecessary to reject the hypothesis that the compared distributions did not suffer.

Thus, these data strongly indicate that the abnormal electroencephalograms are related both to the selected factors in the family history and to severe illness and/or cerebral injury sustained early in life. The lack of significance in the difference between the categories containing illness and/or injury and the category without any positive factors for the patients with primary behavior disorders may be due to either or both of two factors: (1) the high percentage of abnormal electroencephalograms in the category without positive factors, and/or (2) the selective effect of illness or injury on the abnormality of the electroencephalogram.

Since there seems to be a relation between electroencephalographic abnormality and either positive family history or previous severe illness and/or cerebral injury or both, further problems appear. Are certain of the selected criteria for a positive family history or for the personal history of illness or injury of the patient closely related to electroencephalographic abnormality, and are certain others not? The data pertaining to the first part of this question may now be considered. Table 4 presents a summary of the electroencephalographic data in relation to the four selected components of the category of positive family history only: epilepsy, maladjusted personality, chronic alcoholism and psychosis. The category of positive family history and previous illness and/or injury was omitted so that the complications from illness and injury would be minimized. There were 70 patients—25 with primary behavior disorders and 45 with psychopathic personality. Some patients had two or more components in their family history; thus

TABLE 4.—Analysis of the Electroencephalogram with Relation to Factors of the Category of Positive Family History Only

Family History	Primary Behavior Disorders			Psychopathic Personality			Totals		
	Abn *	QN	N	Abn	N	Totals	Abn	QN	Totals
Epilepsy.....	3	0	0	2	0	2	5 (100%)	0	0
Maladjusted personality	8	4	4	19	9	28	27 (61.3%)	4 (9%)	13 (30.7%)
Alcoholism.....	5	2	3	9	8	17	14 (51.9%)	2 (7.4%)	11 (40.7%)
Psychosis.....	4	3	2	6	10	16	10 (40%)	3 (12%)	12 (48%)
No positive family history or previous illness and/or injury....	8 (40%)	1 (5%)	11 (55%)	8 (33.3%)	16 (66.7%)	24 (100%)	16 (38.5%)	1 (2.5%)	27 (62%)
									44 (100%)

* In this table and in tables 5 and 6, Abn indicates abnormal; QN, questionably normal, and N, normal electroencephalograms.

the total number of components will be more than the number of patients involved.

Although there were but 5 patients with a family history of epilepsy, it was of interest to note that the patients coming from those families had abnormal electroencephalograms.

The component that occurred most frequently in the family histories of these patients was maladjusted personality. There were 44 families which contained one or more members so classified. Twenty-seven of these members, or 61.3 per cent, had abnormal electroencephalograms. When the distribution of this group was compared with the distribution of the group without any positive factors by the method of χ^2 , a level of confidence of 1 per cent was found. This means that the difference in the two distributions could be attributed to chance in only 1 out of 100 experiments and is thus statistically significant.

There were 27 patients whose family history was characterized by one or more members being judged as chronically alcoholic. Fourteen patients, or 51.9 per cent, had abnormal electroencephalograms. There were 25 patients whose family history was characterized by one or more members being or having been psychotic. Ten patients, or 40 per cent, had abnormal electroencephalograms. When the distribution of either of these two groups, patients with a family history of chronic alcoholism and patients with a family history of psychosis, were compared by the χ^2 test with the distribution for the group without any positive factors in the family history, the difference was found to be statistically insignificant.

These data, then, suggest that two groups of patients, those in whose family history there were epilepsy and maladjusted personality, respectively, were characterized by a significantly greater proportion of electroencephalographic abnormality than the group with a negative family and personal history. In contrast, the other two groups, patients with a family history of chronic alcoholism and psychosis, were not so characterized.

Since the groups with the family histories of maladjusted personality and chronic alcoholism were logically so similar, yet differed in their comparison with the group with a negative history, they were themselves compared with the χ^2 test. The level of significance attained was definitely insufficient to warrant rejecting the hypothesis that they differed except by chance.

Inasmuch as alcoholism, in the present histories, occurred predominantly in the male parent, and maladjusted personality occurred almost equally in the male and in the female parents, it seemed possible to assess the relationship between the sex of the parent and the abnormality of the patient's electroencephalogram. Of the 44 patients with a positive family history only, with one or more relatives considered as

having maladjusted personality, the father was the closest relative of 15, and the mother, of 17. Of the patients whose fathers were considered as maladjusted, 6 (40 per cent) had abnormal electroencephalograms, while of the patients whose mothers were so considered 13 (76 per cent) had abnormal electroencephalograms. The χ^2 test revealed a level of significance between 2 and 5 per cent. The sex of the parent thus seems related to the abnormality of the patient's electroencephalogram.

Of the 27 patients with one or more relatives who were considered alcoholic, the father was the closest relative of 16, and the mother, of 1. Seven (44 per cent) of the patients whose fathers were judged to be alcoholic had abnormal electroencephalograms. This number was approximately the same as that of patients with abnormal electroencephalograms whose fathers were judged as maladjusted. There were 3 patients both of whose parents were considered maladjusted and 1 patient both of whose parents were considered alcoholic. All 4 patients had abnormal electroencephalograms, thus suggesting that when both parents were involved there was a greater probability of abnormal electrocortical activity in the patient. These patients were too few for statistical analysis.

Further evidence of these relations may be obtained by analyzing the category of patients with positive family history and previous illness and/or injury. Of the 10 patients whose fathers were considered to have maladjusted personality, 5 (50 per cent) had abnormal electroencephalograms. Of the 10 patients whose fathers were considered alcoholic, 6 (60 per cent) had abnormal electrocortical potentials. Of the 6 patients whose mothers were considered to have maladjusted personalities, 4 (66.7 per cent) had abnormal electroencephalograms. There were no patients in this category whose mothers were judged alcoholic. There were 3 patients both of whose parents were considered maladjusted. Two (66.7 per cent) of these had abnormal electroencephalograms. While there were few cases for analysis, the trend for this category was similar to the trend for the category of patients with positive family history only.

When these categories are combined and the patients whose relatives had either maladjusted personality or chronic alcoholism are considered as one group, the data may be summarized as follows: Of 51 patients whose fathers were considered either as having maladjusted personality or chronic alcoholism, 24, or 47 per cent, had abnormal electroencephalograms. Of 24 patients whose mothers were considered to have a similar condition, 17, or 70.8 per cent, had abnormal electrocortical potentials. The χ^2 test revealed a level of significance of 5 to 7 per cent. Of 7 patients with both parents similarly classified, 6, or 85.7 per cent, had abnormal waves. It would thus seem that the incidence of abnormal electroencephalograms would be greater not only

for those patients whose mothers, in contrast to the fathers, were judged maladjusted but that it would be still greater for those patients both of whose parents were so judged. This is strongly suggestive of a relationship between sex of the parent and abnormality in the patient.

The data presented in table 3 indicated that the abnormal electroencephalograms of the patients with psychopathic personality and primary behavior disorders when combined were related to the factors in both the family history and the personal history. To be considered now is the relation of the patients' electroencephalograms to the factors of previous severe illness and cerebral injury. Table 5 presents such a relationship in the category of severe illness and/or cerebral injury only. By considering this category alone, the complication of factors of a positive family history is reduced. The six selected factors in the personal history were anoxia, convulsions, head injury, severe illness, questionable birth injury and prematurity. There were 30 patients with primary behavior disorders who had personal histories which included a total of 47 of these selected factors. There were 17 patients with psychopathic personality who had histories which included a total of 19 factors. Thus, some of the patients had a history of more than one of these factors.

The incidence of a history of convulsions, severe illness and questionable birth injury was greater for patients with primary behavior disorders than for those with psychopathic personality. The numbers for each factor, however, were too small to allow statistical comparisons.

There were 7 patients with a history of convulsions; 5, or 71.4 per cent, had abnormal electroencephalograms. There were 9 patients with a history of head injury; 6, or 66.7 per cent, had electroencephalographic abnormality, of which 3 showed signs of localization. There were 33 patients with a history of severe illness; 21, or 63.6 per cent, had abnormal electrocortical potentials. When the distribution of this last group was compared with the distribution of the category with no positive factors by the χ^2 test, a level of significance of 2 per cent was obtained. There were 14 patients with a history of questionable birth injury; 7, or 50 per cent, had abnormal electroencephalograms. When the distribution of this group was compared with the distribution of the category with no positive factors by the χ^2 test, a level of significance of 50 to 70 per cent was obtained. The first statistical comparison may be regarded as strongly suggesting that the two distributions differ, while the second does not.

The largest group, that of 33 patients with previous severe illness, lent itself to further analysis. Table 6 presents an analysis of the electroencephalographic data with reference to the degree of severity and the age of occurrence of the illness for both the category of severe illness only and that of positive family history and severe illness. In the category of severe illness alone only 30 patients could

TABLE 5.—Analysis of the Electroencephalogram with Relation to the Factors of the Category of Severe Illness and/or Cerebral Injury Only

Illness or Injury	Primary Behavior Disorders			Psychopathic Personality			Totals		
	Abn	QN	N	Abn	QN	N	Abn	QN	N
Anoxia.....	0	0	0	0	0	0	0	0	0
Convulsions.....	4	0	2	1	0	0	5 (71.4%)	0	2 (28.6%)
Head injury.....	3	0	2	3	1	1	6 (66.7%)	0	3 (33.3%)
Severe illness.....	15	0	8	6	4	4	21 (63.6%)	0	12 (36.4%)
Birth injury.....	5	0	6	2	1	3	7 (50.0%)	0	7 (50.0%)
Prematurity.....	1	0	1	0	0	1	1 (33.3%)	0	2 (66.7%)
No positive family history or previous illness and/or injury.....	8 (40.0%)	1 (5.0%)	11 (55.0%)	8 (33.3%)	16 (66.7%)	24 (100%)	16 (36.5%)	1 (2.5%)	27 (62.0%)
									44 (100%)

TABLE 6.—Analysis of the Electroencephalogram with Relation to the Degree of Severity* and Age of Occurrence of Illness

Degree of Severity	1 to 3 Years			4 to 6 Years			7 Years			Totals		
	Abn	QN	N	Abn	QN	N	Abn	QN	N	Abn	QN	N
Severe illness only.....	5	0	0	1	0	0	1	0	0	7 (100%)	0	0
++	7	2	5	3	0	5	1	5	5	11 (47.8%)	0	12 (52.2%)
Totals.....	12	2	5	4	0	5	2	5	5	18 (60.0%)	0	12 (40.0%)
Positive family history and severe illness.....	3	0	0	1	0	2	0	0	0	4 (66.7%)	0	2 (33.3%)
++	3	3	3	2	1	1	3	1	1	8 (57.1%)	1 (7.9%)	5 (35.7%)
Totals.....	6	3	3	3	1	3	3	1	1	12 (60.0%)	1 (5.0%)	7 (35.0%)
++	8	0	2	2	0	2	1	0	0	11 (84.0%)	0	2 (15.4%)
Totals.....	10	5	5	5	1	6	4	6	6	19 (51.8%)	1 (2.8%)	17 (45.9%)
Totals.....	18 (78.3%)	5 (21.7%)	7 (41.2%)	1 (8.8%)	8 (50.0%)	5 (45.5%)	6 (54.5%)	30 (60.0%)	1 (2.0%)	30 (60.0%)	1 (2.0%)	19 (38.0%)

* Very severe illness is indicated by ++++; moderately severe illness by ++.

be used, for the age at which the other 3 patients were ill was not known. The degree of severity was subdivided into the very severe (+ + + +) and the moderately severe (+ +). There were 7 patients with very severe illnesses: Four had prolonged elevation of temperature, signs of meningeal irritation and coma, followed by a history of temporary regression in development; 2 had prolonged high temperatures with prolonged delirium or coma, and 1 had a severe attack of pertussis at the age of 6 months. The moderately severe illnesses were all characterized by high temperatures and stupor or delirium but no meningeal irritation or history of subsequent regression in development. All patients who had very severe illnesses only had abnormal electroencephalograms. Eleven of the 23 patients with histories of moderately severe illness only had abnormal electroencephalograms.

In the other category, that of positive family history and severe illness, there were 6 patients with a history of very severe illness, 4 (66.7 per cent) of whom had abnormal electroencephalograms, and 14 patients with moderately severe illness, 8 (57.1 per cent) of whom had abnormal electroencephalograms. The consistency of the data for these two categories, then, would seem to indicate that severity of illness is related to the development of an abnormal electroencephalogram.

When the distribution for very severe illness, regardless of family history, was compared with the distribution for moderately severe illness, regardless of family history, by means of the χ^2 test, the level of significance was between 2 and 5 per cent.

Further examination of the table indicates that the age at which the illness occurred seems to bear some relationship to the electrical activity of the brain. It may be noted that, irrespective of the degree of severity of the illness, the younger the patient at the time of the illness the greater the probability of his having an abnormal electroencephalogram. Of those patients whose illnesses occurred within the first three years of life, 78.3 per cent had abnormal electroencephalograms. For those whose illnesses occurred after that time, only 44.4 per cent had a similar abnormality. Although the data are not presented, the type of illness, such as pneumonia, pertussis, measles and fevers of unknown origin, seemed to show no relation to the type of the electroencephalogram; neither did the time interval between the occurrence of the illness and the electroencephalographic recording. Thus, two factors seemed to be operating, more or less independently, in association with a severe illness to produce long-lasting disturbance in the electrical activity of the brain: the degree of severity of the illness and the age at which the illness occurred.

COMMENT

The diagnoses of primary behavior disorders and psychopathic personality refer primarily to the personality structure and the behavior on

a descriptive basis rather than to any constellation of factors important in causation of the disturbances. They are descriptive diagnoses which most probably include a number of clinical conditions. The data presented in this report suggested this. The two diagnostic groups in this study were characterized by a high incidence of electroencephalographic abnormality. The higher incidence of paroxysmal electroencephalograms in the patients with primary behavior disorders than that in the patients with psychopathic personality would imply that more patients with preclinical epilepsy were included in the former than in the latter group. It is to be expected that the latter group would contain fewer such patients, for they were older and age acted as a selective factor. Moreover, the greater incidence of severe illness or cerebral injury sustained early in life in the younger group than in the older one would suggest these factors, too, as possibly etiologic. Thus, within the large descriptive diagnostic groups there may be, on the basis of the data presented, smaller, more specific etiologic subgroups.

The data strongly suggest that abnormal electroencephalograms were obtained for a higher percentage of patients who had either a positive family history or a personal history of antecedent severe illness or cerebral injury than for those patients for whom neither factor appeared. In the family history, the factors of maladjusted personality and epilepsy appeared of greater importance than the factor of psychosis. The designation of "alcoholism" really included disturbance in personality function. When the histories were analyzed to discover whether the male or female parent showed maladjustment or alcoholism, it was found that there was a higher incidence of abnormal electroencephalograms among the patients whose mothers were evaluated as maladjusted or alcoholic than in those patients whose fathers were so judged. This relationship is consistent with the data reported by Lennox, Gibbs and Gibbs¹⁰ on the inheritance of cerebral dysrhythmia and epilepsy. Their data, too, suggested the possibility that females carry a greater inheritance factor, as expressed by dysrhythmia and by family history, than that carried by males. As in their data, so in ours, there is no suggestion of sex linkage in the occurrence of dysrhythmia.

Lennox, Gibbs and Gibbs¹⁰ stated that approximately 60 per cent of the near relatives of epileptic patients had abnormal electrocortical activity. However, only 2.4 per cent (parents, siblings and children) had a history of seizures. Thus, persons with abnormal electroencephalograms outnumber persons with seizures in a ratio of 25:1. Since the incidence of epilepsy in the population is 0.5 per cent, they concluded

10. Lennox, W. G.; Gibbs, E. L., and Gibbs, F. A.: Inheritance of Cerebral Dysrhythmia and Epilepsy, *Arch. Neurol. & Psychiat.* **44**:1155-1183 (Dec.) 1940.

that approximately 12 per cent of the population have an abnormal electroencephalogram—"about 15,000,000 in the United States"—a statement supported by random electroencephalographic sampling of the population. The samples reported here of the population with psychopathic personality and primary behavior disorders were characterized by high incidences (58 and 56 per cent, respectively) of abnormal electroencephalographic activity. It is well known that many relatives of epileptic patients who do not have epileptic manifestations reveal unusual behavior patterns. One may therefore ask: What is the relation of the populations under study here to the seizure-free relatives of epileptic patients? What is the relation of these populations to the 15,000,000 persons in the United States with cerebral dysrhythmia?

Although the data would indicate a relation of the abnormal electroencephalogram to psychopathic personality or primary behavior disorders, it must not be considered pathognomonic for either. This is obvious from the electroencephalographic examination of any psychiatric or neurologic population. In fact, Liberson and Seguin¹¹ have indicated a relation between a positive family history and abnormal electrocortical activity for a number of psychiatric conditions, with the inference of inheritance of the abnormal waves. Moreover, there are persons generally conceded to be "normal" who show electroencephalographic characteristics falling beyond the present concept of normality. This is of common electroencephalographic experience: It has been reported¹⁰ for relatives of patients with epilepsy, and it is known to be true for relatives of patients with either psychopathic personality or primary behavior disorders.¹² Abnormality of the electroencephalogram is not necessarily indicative of abnormality in behavior; it is merely indicative of an apparently reliable probability that there may be some kind of behavioral deviation sooner or later. This may account for the relatively high percentage of abnormal electroencephalographic activity in the group of patients without either a positive family history or a personal history of severe illness or cerebral injury sustained early in life.

This relatively high percentage of abnormal electroencephalograms in the group of patients without any positive abnormality in the family history or personal history of illness or injury for both primary behavior disorders and psychopathic personality is most probably inherited.¹² This probability must be kept in the foreground when considering the factors of severe illness and cerebral injury. An evaluation of any

11. Liberson, W. T., and Seguin, C. A.: Brain Waves and Heredity in Mental Diseases, *Psychosom. Med.* 7:35-38 (Jan.) 1945.

12. Unpublished data on the relationship of the electroencephalograms of the parents to those of the patients.

experience of the latter kind is complicated by its possible superimposition on the inherited characteristics of the patient populations. Nevertheless the patients who had personal histories of convulsions, head injury with unconsciousness or severe illness showed a higher incidence of abnormal electrical activity of the brain than the patients with no such experiences. Factors of severe illness which were of importance were the degree of severity and the age at which the illness occurred. The more severe the illness and the younger the patient at the time of its occurrence, the greater the probability of an abnormal electroencephalogram. This was true irrespective of the type of the severe illness. Heppenstall and Hill¹³ have likewise shown that patients under 20 years of age at the time of a head injury had a greater probability of abnormal electrocortical activity than those who were older. A higher incidence of abnormal electrocortical activity in patients having a history of convulsions during early life is to be expected. The probability of their relation to epilepsy is greater than that due to chance.

The implications of an abnormal electroencephalogram are clear—that there probably is some pathophysiologic process in the cortex. The implications of a normal record are equally clear—that there probably is no pathophysiologic process in the cortex. These implications must be considered in terms of the theory of probability, for it is known that such a process may be dormant at the time of recording, and thus not be made apparent on the record. Furthermore, assumptions of the presence or absence of a pathophysiologic process can at present be referred only to the cortex. Subcortical processes may not be normal; yet the electroencephalogram may be repeatedly without discernible abnormal activity.

The electroencephalographic data, therefore, would indicate that there is a high incidence of discoverable and repeatedly verifiable abnormal organic processes in the heterogeneous group of disturbances diagnostically categorized, respectively, as primary behavior disorders and psychopathic personality. These abnormal processes would appear to be inherited. Furthermore, pathophysiologic experiences, either chemogenic or histogenic, if either early enough or severe enough, would likewise lead to the development of abnormal processes. May not the inference be made that these abnormal processes are related to the maladjustment of the patients, that they may indicate the organism's susceptibility to difficulties in behavioral adjustment, that persons with such abnormal processes may possess less elasticity in their neural limits for withstanding the stresses and strains of the adjustment process,

13. Heppenstall, M. E., and Hill, D.: Electroencephalography in Chronic Post-Traumatic Syndromes, *Lancet* 1:261-263 (Feb. 27) 1943.

that their response tendencies have been altered and that they have poorer adaptation in their interreaction with the social environment?

These abnormal organic processes should not be considered the sole etiologic factor for the behavioral disturbances of the patients. Etiology from a psychiatric viewpoint always involves a constellation of factors, genogenic, histogenic, chemogenic and psychogenic, with their relative interrelationships. The presence of an abnormal electroencephalogram would add weight to genogenic, histogenic and chemogenic factors, which otherwise might be undervaluated. The majority of the abnormal electroencephalograms would appear to be genogenic; a minority would appear to be physiogenic (histogenic or chemogenic); for normal electroencephalograms no inferences as to disturbed pathophysiologic cortical processes could be made, although such may be present. The electroencephalogram cannot, of course, indicate the totality of the etiologic constellation, although it may assist in the more adequate evaluation of the etiologic factors in a given patient's disturbance: ancestral transmission, physical trauma and social or emotional trauma.

SUMMARY

1. Two hundred patients, 100 each with primary behavior disorders and with psychopathic personality, showed considerably higher percentages of electrocortical abnormality, 56 and 58 per cent, respectively, than the percentages reported for presumably neurologically normal children and adults.
2. Fourteen per cent of the patients with primary behavior disorders, in contrast to 2 per cent of the patients with psychopathic personality, had paroxysmal electroencephalographic activity.
3. The incidences in the family history of epilepsy, maladjusted personality, chronic alcoholism and psychosis were similar in the two diagnostic groups.
4. The incidences in the personal history of convulsions, severe illness and questionable birth injury were greater for the group of patients with primary behavior disorders than for the group with psychopathic personality. The incidences of head injury were similar for the two groups.
5. When the two groups of patients were combined, significantly greater proportions of abnormal electroencephalograms were found when there was a family history either of epilepsy or of maladjusted personality.
6. The proportion of patients showing electroencephalographic abnormality appeared to be greater when the mothers were judged maladjusted or alcoholic than when the fathers were so judged.

7. When the two groups of patients were combined, significantly greater proportions of abnormal electroencephalograms were found when there was a personal history of convulsions, head injury with unconsciousness or severe illness.

8. For the category of severe illness, the younger the patient at the time of the illness and/or the more severe the illness, the greater the probability of abnormal electrocortical activity.

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CLEIDOCRANIAL DYSOSTOSIS WITH PSYCHOSIS

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THERE was recently admitted to and treated at Duke Hospital a patient with cleidocranial dysostosis and a psychosis. The case was of especial interest because of the concatenation of the rare anatomic anomaly and schizophrenia. Particularly noteworthy in this case was the absence of psychotic manifestations in the patient's father, who possessed essentially the same physical defects to an even greater degree. The rarity of cleidocranial dysostosis, the constancy of the physical manifestations of the syndrome and the autosomal mendelian dominant mode of inheritance of the trait in at least the majority of instances¹ make it worth while to redescribe this condition and to report an additional case even if it were not for the unusual psychiatric implications.

Cleidocranial dysostosis is a rare syndrome, affecting persons of both sexes and of all ages, and occurs in diverse racial and national groups. In 1760 Meckel² reported the case of an infant born without clavicles, and in 1765 Martin³ gave details of a family several members of which had abnormal clavicles. In 1897 Marie and Sainton⁴ described the association of clavicular and cranial defects in members of two families, and the following year they proposed the name *dysostose cléido-crânienne héréditaire*.⁵ In a monograph Hultkrantz⁶ discussed

1. Lasker, G. W.: The Inheritance of Cleidocranial Dysostosis, Human Biol., to be published.

2. Meckel: Mém. de Paris, 1760; cited by Terry, R. J.: Rudimentary Clavicles and Other Abnormalities of the Skeleton of a White Woman, J. Anat. & Physiol. **33**:413-422, 1899.

3. Martin: Sur un déplacement naturel de la clavicule, J. de méd., chir., pharm. **23**:456-460, 1765.

4. Marie, P., and Sainton, P.: Observation d'hydrocéphalie héréditaire (père et fils) par vice de développement du crâne et du cerveau, Bull. et mém. Soc. méd. d. hôp. de Paris **14**:706-712, 1897.

5. Marie, P., and Sainton, P.: La dysostose cléido-crânienne héréditaire (hydrocéphalie héréditaire), Bull. et mém. Soc. méd. d. hôp. de Paris **15**:436-437, 1898.

6. Hultkrantz, J. W.: Ueber Dysostosis cleido-cranialis: kongenitale kombinierte Schädel- und Schlüsselbeinanomalien, Ztschr. f. Morphol. u. Anthropol. **11**:385-528, 1908.

all the material available in 1908, and this publication remains one of the most thorough treatises on the subject. A good general description in English is provided by Eldridge, Simon and Ramos,⁷ and a modern review is given by Carrière and associates.⁸ In 1929 Fitchet⁹ published an excellent review, with a bibliography of over 125 items and with abstracts of most of the cases. Engel,¹⁰ in a systematic study of the syndrome, summarized 228 cases and noted that 87 additional cases were said to have occurred in the families of the affected patients. We have used some of Engel's statistics on cases published up to 1933, inclusive, in compiling our summaries. Since 1933, 145 new cases have been reported and an additional 63 cases mentioned. Altogether, there have been approximately 275 publications on the subject, of which 245 have been reviewed by one of us (G. W. L.).¹¹ However, the infrequency of the condition is attested to by the fact that only 1 previous case of cleidocranial dysostosis appears in the records of the quarter of a million patients seen at Duke Hospital since its opening, fifteen years ago.¹²

In a typical case of cleidocranial dysostosis the head is large, especially in comparison with the face; and it is wide and flat, with protruding cranial bosses and bulging frontal region. The bones of the cranial vault are slow to develop, so that the fontanels and sutures are widely separated at birth and may remain apart into adulthood. Usually there is a depression at the fontanels, and sometimes a pulse may be felt there if the bone is absent. There are furrows along the lines of the sutures, which mark thinning or separation of the bones. The bosses, however, are much thickened. One of the most conspicuous clinical signs is a sagittal groove dividing the forehead and marking the frontal suture, which regularly persists in cases of this anomaly. Many wormian bones occur in the sutures, and extra sutures have been observed dividing the parietal bones (horizontally) and the malar bones and occurring elsewhere. The frontal sinuses have been reported

7. Eldridge, W. W.; Simon, A., and Ramos, R.: Cleidocranial Dysostosis: A Case Report, *Am. J. Roentgenol.* **34**:41-49, 1935.

8. Carrière, G.; Huriez, C., and Décamps, G.: La dysostose cléidocrânienne (maladie de Pierre Marie et Sainton), *Gaz. d. hôp.* **110**:701-707, 733-741 and 765, 1937.

9. Fitchet, S. M.: Cleidocranial Dysostosis: Hereditary and Familial, *J. Bone & Joint Surg.* **11**:838-866, 1929.

10. Engel, E.: Dysostosis Cleidocranialis, *Helvet. med. acta* **4**:158-174, 1937.

11. A complete bibliography will be found in conjunction with Lasker's article.¹

12. Dr. Angus McBryde has permitted us to examine the records, photographs and roentgenograms in a case of cleidocranial dysostosis in a 12 year old white boy, who had typical cranial and clavicular anomalies and pronounced pelvic changes, including a very wide pubic symphysis. This boy was of normal intelligence and the only child of normal parents.

to be absent or much reduced in 24 cases. Supraorbital ridges are frequently absent. The mastoids are often virtually lacking, a trait which has been explained as a result of absence of the clavicles and consequent maldevelopment of the sternocleidomastoid muscles. However, a case has been reported by Rhinehart¹³ in which the mastoids were missing but the clavicles were normal. The base of the skull is frequently poorly developed (which may contribute to the brachycephaly), and platybasia, with downward bending of the occiput, is mentioned in some cases. The sella turcica was noticed to be large in 5 cases but was reported as normal in 16 other cases and as small in 2 cases.

The face is usually poorly developed. The bridge of the nose is cartilaginous, the nasal, and sometimes the lacrimal, bones being deficient or completely absent. The root of the nose is therefore regularly depressed, but it is often wide, so that a mongoloid fold may cover the inner canthus of the eye. Hypertelorism has been reported in 17 cases. The orbits are relatively high, but a tendency to exophthalmos is sometimes reported. The malar bones are small, and the zygomatic arch in some cases is incomplete. The palate is typically high-arched and narrow, and development may be defective, leaving a cleft. In 20 cases the high palate was lacking, but even in several of these the palate was narrow. The mandible, being usually prognathous, is frequently disproportionate to the rest of the face. This may be caused chiefly by maldevelopment of the cranial base and a correlated underdevelopment of the middle of the face. In several cases the mental symphysis has been reported to persist.

The dentition is usually conspicuously affected. The milk teeth appear later than is normal in infants but are usually not anomalous in other respects. They are not replaced at the proper time by the permanent teeth, and there is virtually never a full complement of teeth in the mouth of the adult, despite a tendency to supernumerary anterior teeth. Unerupted teeth are found in the mandible and maxilla in most cases, and eruption of teeth in older persons is frequently noted. Dentigerous cysts sometimes occur. The teeth are usually irregular in position, and the occlusion is so bad that many persons have their teeth extracted and wear dentures. Advanced dental caries is often reported. Enamel hypoplasia, malformed roots and enlargement or dwarfing of teeth are sometimes seen. Speech defects associated with the dental anomalies have been reported in 3 cases.

In the case reported by Russo-Frattasi¹⁴ there was an abnormality of the right middle ear and of the tympanic membrane. In the case

13. Rhinehart, B. A.: Cleidocranial Dysostosis (Mutational Dysostosis) with a Case Report, *Radiology* **26**:741-748, 1936.

(Footnotes continued on next page)

of Eldridge and others⁷ there was a history of partial deafness for twenty-two years. Stahl's¹⁵ patient was partially deaf, and 2 of the dysostotic members of the family studied by Miles¹⁶ were deaf.

The dysostotic person is often very short. For 34 men for whom the stature was reported, the height averaged 156.6 cm. and ranged from 132 to 178 cm. Among 34 adult women, the recorded heights ranged from 110 to 159 cm., with an average height of 144.6 cm. Medium statures are found occasionally and probably occur more frequently than is stated in the published reports. Altogether, 131 patients were described as small or slender, 32 as of average size and only 1 as large. In children, as in adults, dwarfing may be very common with this condition. In infants, however, the size is frequently normal, and the stunting becomes apparent only later.

The short stature is usually, at least in part, the result of abnormal curvatures of the vertebral column, kyphosis, lordosis or scoliosis being mentioned in about 55 cases. Scoliosis is the most frequent. Wedging of the vertebral bodies is common. Spina bifida occulta has been reported in 51 cases in which roentgenograms were made and often occurs in the cervical and upper dorsal regions (which is uncommon in the general population). At least 3 instances of cervical meningocele have been reported. The thorax is sometimes conical or funnel shaped and sometimes flattened from side to side. Many cases with a rachitic appearance of the thorax or ribs have been reported. The sternum was depressed in several cases, and the manubrium and the xiphoid process have been found lacking. Cervical ribs have been reported in 7 instances.

Defective clavicles constitute the most characteristic trait of cleidocranial dysostosis. One or both bones may be completely lacking (10 and 30 cases, respectively), but more frequently one or both are imperfect. Frequently there is a sternal bony rudiment, or even an only slightly shortened bone attached to the sternum. Occasionally there is also an acromial rudiment, which may be widely separated from the other fragment or may form a pseudoarthrodial joint. Especially when one only of the clavicles is divided (the right clavicle remained intact in 5 cases and the left in 27), the differential diagnosis of an ununited fracture may be of practical importance. One patient with a unilateral congenital clavicular defect is reported to have swindled thousands of

14. Russo-Frattasi, G.: Rara anomalia dell'orecchio medio in disostosi cleidocranica, Oto-rino-laring. ital. **4**:484-502, 1934.

15. Stahl, F. C.: Neue Beiträge zur Physiognomik und pathologischen Anatomie der Idiotia endemica (genannt Cretinismus), Erlangen, Ferdinand Enke, 1848, pp. 53-57.

16. Miles, P. W.: Cleidocranial Dysostosis: Survey of Six New Cases and One Hundred and Twenty-Six from the Literature, J. Kansas M. Soc. **41**:462-468, 1940.

dollars by feigning a fracture. Another practical problem arose in a case in which surgical removal of a clavicular fragment was required to relieve symptoms caused by pressure on the brachial plexus, and in a similar case a bone graft was employed to reunite the free ends of the clavicle.

In a few persons related to patients with the typical syndrome one finds clavicular anomalies but no alteration of the skull. The signs referable to the skull are therefore not necessarily always present with the dysostosis. On the other hand, in a few other close relatives of persons with typical cleidocranial dysostosis characteristic cranial symptoms have been reported as associated with normal clavicles. Since the clavicles may sometimes be normal, Fitchet's⁹ name "cleidal dysostosis" is inadequate to cover all instances properly belonging to the syndrome.

Other defects of the shoulder girdle have been recorded. The scapula is frequently small, and the superior fossa and the coracoid process may be absent or reduced. Anomalies of the musculature have been variously described in numerous cases, but these are apparently secondary to the lack of clavicles. There are occasional anomalies of the humeral head, and 7 instances of congenital dislocation of the humerus have been reported.

The most prominent symptom is the unusual motility of the shoulders, which in some cases can be voluntarily approximated in front. At least 1 man with such a condition made his living in the circus as a contortionist; another was a ballet dancer. There is usually no functional loss in the arms. Many such persons do heavy work, and 1 youth has been photographed standing on his hands. In 6 cases weakness in the arms is mentioned; in 9 cases there was pain in the arm or shoulder, and in 4 cases paresthesias occurred in the arm.

The pelvic girdle frequently is affected. The most common pelvic anomaly, a wide symphysis pubis, is mentioned in 33 of the cases reported since 1933. The pelvic inlet may be unduly narrow and distorted (24 cases). Eleven cases in which cesarean section was necessary have been reported.

Anomalies of the legs, to which some of the reduction in stature may be ascribed, are extremely common. The literature includes at least 47 cases of genu valgum, 27 cases of coxa vara, 13 cases of pes valgoplanus and a few cases each of many other types of deformity of the lower extremities. Several patients are said to have complained of "weak legs," and several were slow in learning to walk. However, of those slow in learning to walk, at least 3 were also said to be slow in learning to talk.

Still¹⁷ noticed shortening of the thumbs, and Jansen,¹⁸ who noted shortening of the intermediate and terminal phalanges of the toes, widening of the latter and shortness of the nails, proposed the name "dysostosis cleido-cranio-(digitalis)." Short, tapering terminal phalanges have been reported in 66 cases of cleidocranial dysostosis. Carpal, tarsal, metacarpal and metatarsal bones are also sometimes abnormal. In several cases there were extra epiphyses at the base of the second metacarpal bones.

There are in various cases all manner of congenital bony anomalies, but any of them may be lacking. Most notable, except for the clavicular dysostosis, are the many midline defects, which range from persistence of the frontal suture to total absence of the anterior thoracic wall, with extrusion of the heart and lung, as in Morand's¹⁹ case. Other midline defects include persistent fontanels; saddle nose; high, narrow palate; failure of mandibular union at the symphysis; os hyoideum bipartum; sternal defects; spina bifida occulta or spina bifida aperta, and *Spaltbecken*.

REPORT OF A CASE

History.—E. McG., a 31 year old, single engineer, was brought to Duke Hospital by his mother because of inability to sleep and "nervousness," of about two months' duration. For the past week he had been "talking out of his head" and had behaved queerly.

The patient's background was that of a well regarded family of moderate means in a small town. His father, aged 72, was described by the patient's mother as alert and "very much alive" despite his pronounced skeletal and dental defects. His case will be described later. Mrs. McG. mentioned that 1 of her husband's sisters and 1 of his brothers were afflicted with "mental trouble." No history of physical defects was obtainable except that the patient's paternal grandfather and the latter's sister were described as stooped in their old age. There were 2 brothers of the patient, both of whom were normal physically and mentally. Both were married and apparently well adjusted. The mother herself seemed to be a serious-minded, hard-working person, who was interested in her children and had always been fairly ambitious for them. She described herself and her husband as always having been only moderately strict with the boys. Both she and her sister were physically normal.

The patient's personal development was interesting in that it was much like that of his brothers except that his enuresis lasted longer, until the age of 4 years. He was at times quite sensitive about his defects but played just like his brothers and stayed no more to himself than they. He studied more diligently than the others, and his mother thinks that possibly he was a little slower. Until he

17. Still: A Case of Cleidocranial Dysostosis, *Tr. M. Soc. London* **31**:350-352, 1908.

18. Jansen, M.: *Feebleness of Growth and Congenital Dwarfism*, with Special Reference to Dysostosis Cleido-Cranialis, London, Oxford University Press, 1921.

19. Morand: *Observations anatomiques*, *Hist. Acad. roy. d. sc.* **4**:476, 1776; cited by Fitchet.⁹

became interested in radio and made a broadcasting station of his own, in which he became absorbed, she thought that he was hard of hearing. He was described as very religious, and his mother hesitated before pronouncing him as not fanatical. There had been no bad habits, such as drinking, smoking or drug addiction.

After graduating from a college of engineering, he was unable to find work as an engineer and seemed to be disturbed and nervous about it. At that time he first came to Duke Hospital to see what could be done for his dental and other difficulties. The diagnosis of cleidocranial dysostosis was made, and he was told that nothing could be done for him. His nervousness seems to have presented no features similar to the disorder responsible for his latest hospitalization. For several years he helped his father, did other odd jobs and for a little while taught school. He remained ambitious to "achieve something big" as an engineer and decided to go to New York. He learned that he had to have a hernia repaired before he could find employment. After the herniorrhaphy he worked for the electrical department of a large railroad, then secured a job with Westinghouse Electrical Corporation in electrical engineering and worked toward his master's degree at Columbia University. He devoted much time to church work and Y. M. C. A. activity. He seemed to like New York, and the only disturbing incident of which his mother knew was the possibility of developments with a girl with whom he had become friendly.

For two months before admission to the hospital the patient had been nervous and had experienced difficulty in sleeping. He stayed away from work for a short time but was advised to return. He became certain that something was wrong with his heart. An electrocardiogram was normal, however. Having been granted a leave of absence, he returned home, and his condition was not "really bad" until a week before his admission to the hospital. He talked about not having known the facts of life, became very nervous, was tense but was fairly quiet. He was never destructive and did not threaten any one. He talked about the cause of his illness and wished some one had helped him. On the day before admission he urinated without going to the toilet and experienced enuresis that night.

On admission he appeared to be antagonistic and apprehensive. He sat wrapped in a heavy robe, perspiring profusely, and his teeth clacked rapidly. There were numerous stereotyped movements, and his speech was barely coherent. He stated that he could not concentrate, that he felt at times like taking his own life and that he was no good, as his genitalia were made of water and his heart was enlarged. He believed that he had just killed his parents, and he was actively hallucinated. He frankly stated that he was afraid of the hospital physicians because he thought that they had already tried to hit him on the head, and he pleaded with them not to do it again. He remarked that he was "just weak" and that he masturbated and could not stop. Since he believed that no one would help him, he would not say whether he thought he could improve, though his attitude implied that he believed his case was hopeless.

Examination.—The patient weighed 61.5 Kg. and was 164 cm. (64½ inches) in height. His head was large and wide, with a sagittal furrow beginning at the root of the nose and extending the length of the crown. The eyes appeared large, and the sclera could be seen on all sides of the iris when the patient looked straight ahead. The nose was short and broad, and the root was flat and marked by a vertical groove, several millimeters wide, in the region of the nasion. The bridge was deficient. The middle part of the face was poorly developed, partly because of the lack of a full complement of teeth, and the mandible was prominent.

The teeth were anomalous. Seven maxillary teeth were fully erupted and two others partially so. Ten mandibular teeth were present. The probable formula was: $\frac{7\ 6\ 4\ 2}{7\ 6\ 3\ 2\ 1} \mid \frac{1\ 2\ 5\ 6\ 7}{1\ 3\ 4\ 6\ 7}$. Because all teeth were extremely irregularly placed, the occlusion was unsatisfactory. The lower left anterior teeth had gaps between them and were rotated. The enamel on the mandibular incisors was defective. The palate was an extremely high, narrowed gothic arch, and the tooth-bearing part was much widened medially. There was a history of pyorrhea in 1935.

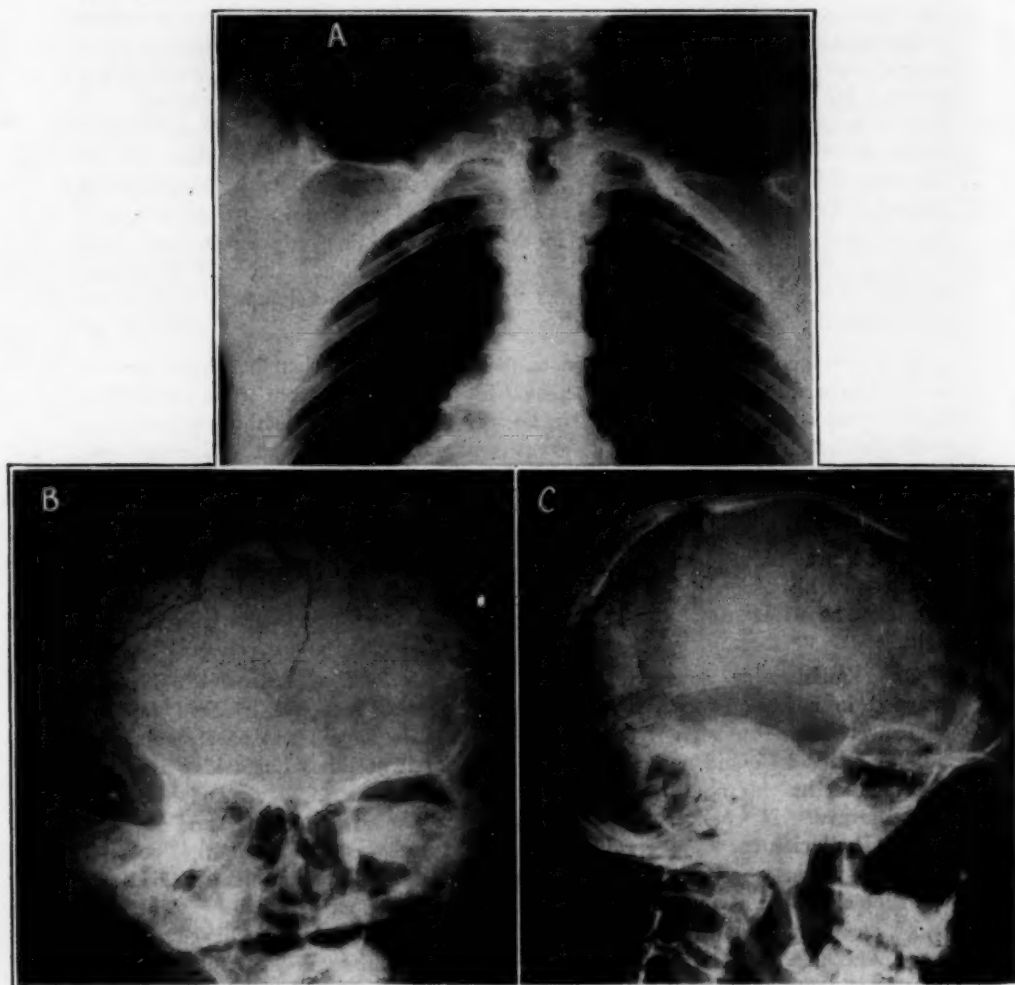


Fig. 1.—(the patient, E. McG.) *A*, roentgenogram of the chest, showing the sternal rudiment of the clavicle and spina bifida; *B*, roentgenogram of the skull (brow down), showing the frontal suture, the reduced frontal sinus, the sutural furrow and defective nasal bones; *C*, lateral roentgenogram of the skull, showing defects of the inner table, impacted third molar and other features.

Roentgenograms of the patient had been taken in the outpatient department in 1935, and the generalized findings of cleidocranial dysostosis with widening of the suture lines were reported. Additional roentgenograms were taken in 1944

(fig. 1), when the curvature of the dorsal region due to the narrowing of the middorsal vertebrae was noted. The general appearance was said to be that of osteochondritis of long standing.

Dr. Max Eichwald, of Watts Hospital, Durham, N. C., subsequently examined the roentgenograms and commented as follows: "The films are not clear. They consist of a lateral and an anteroposterior (brow down) view of the skull, views of both shoulders and of the chest and a lateral view of the vertebral column. They show a cranial cavity which is large in relation to the face, and a scaphoid,

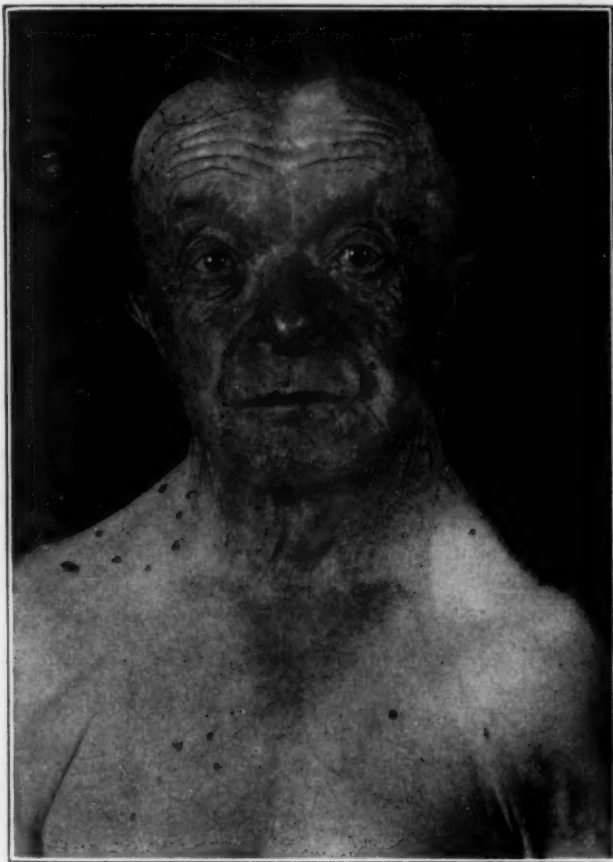


Fig. 2.—Cleidocranial dysostosis in the father (W. McG.), showing absence of clavicles, wide head and frontal furrow.

asymmetric skull with several wormian bones and a wide sagittal suture, not quite closed. Both the inner and the outer table of the skull are present except for interruption of the inner table in the posterior part of the frontal and the posterior part of the parietal region. There are flattening and anteroposterior elongation of the sella turcica. The frontal sinuses are undeveloped. On the left there is a very small nasal bone; none is present on the right. The orbits are high. One mandibular third molar is visible, unerupted and malposed. The left clavicle is represented by a short, underdeveloped sternal rudiment, which does not extend to the acromial process, and the right clavicle seems to be similar but

smaller. The scapulas are only slightly smaller than normal. The upper dorsal vertebral bodies are wedge shaped, producing marked kyphosis. Spina bifida occulta occurs in at least the lowest three cervical vertebrae (no roentgenograms were taken of the upper cervical region). The ribs are narrow. There are no cervical ribs in this case."

Neurologic examination revealed nothing significant so far as could be ascertained with poor subjective testing. The electroencephalogram, the serologic reactions and other accessory clinical findings were essentially normal.

Patient's Father.—W. McG., the patient's father, was physically similar to his son. He is pictured in figure 2, and in figure 3 he is shown with the 2 normal



Fig. 3.—Cleidocranial dysostosis in the father and his normal sons. The third (affected) son, E. McG., is not shown.

brothers of the patient. He was 132 cm. (52 inches) tall. Physical examination revealed the absence of clavicles and widening of the neck. The furrow of the forehead and crown and the wide cranial vault were typical of cleidocranial dysostosis. The fingers, especially the fifth, showed deficient growth of the terminal phalanges. An inguinal hernia was present. The teeth were all extracted about thirty years before. W. McG said that he kept cutting teeth until he was 44 years old, and one upper right molar appeared still to be erupting at the time of his examination. He said he had had seventeen impacted teeth. The palate appeared normal in shape for an edentulous person. He was hard of hearing.

A summary of the readings of the roentgenograms of W. McG. is as follows: The thorax was narrow at its superior aspect, and the clavicles were completely absent. The roentgenogram of the skull showed that the anteroposterior and transverse diameters were increased, with the vertex flat and with wide separation of the frontal bones. In the occipital area there was inward protrusion in the region of the foramen magnum, simulating actual platybasia. The changes in the chest and skull were those of cleidocranial dysostosis.

Course of Patient's Illness.—After a few days at the hospital, the patient, E. McG., became mute and would respond only to whispered communications. At this time he had three interviews when under sodium amytal hypnosis, each of which produced successively poorer results. During the first session he talked much more freely than at any other time during his stay in the hospital. He spoke about the F.B.I. and charges against him. He talked about his girl and indicated sensitivity concerning his physical defects in interpersonal relationships, particularly with this girl. He also talked at length, though vaguely, of participating under force in homosexual activities. He believed that he had killed his parents and remarked that he had seen the holes in his mother's legs. It was interesting that his active hallucinations disappeared during the administration of amytal and that when the effects of the drug began to wear off the hallucinations returned.

As was expected, no improvement resulted from the interviews during amytal hypnosis. For an hour or more every evening of the subsequent two or three weeks efforts were made to obtain contact with the patient. Much of the time was spent in waiting. Occasionally there appeared to be sparks of interest in some subject, but nothing came even close to taking hold of his attention. All the psychogenic material available was gradually used, with no noticeable results, and it was decided to proceed with electric shock treatment, with use of curare because of his vertebral condition; a series of seventeen electric shocks was given. The first four treatments brought a great deal of improvement, but he rapidly relapsed. Then followed four more treatments at closer intervals, the response to which was only moderately favorable. After another short period, another nine treatments were given, but never did he reach the state of clarity that he had attained after the first four treatments. He was somewhat confused, and it was decided to give him a trial at home.

Nineteen months later the patient was seen briefly at his home. He said that he had been hospitalized elsewhere in the meantime and that he had received more electric shock treatments.

The patient's mother has died recently, and at the time of this report he lives alone with his father. He has not been working but seems to be able to maintain normal social relations with the neighbors. However, he admitted having auditory and visual hallucinations and said that he slept only about three hours a night. He claimed that he could be influenced from afar, that he had been killed and that he will be killed. He does not trust any one, not even his father or brothers.

A review of the literature suggests that from the psychiatric standpoint persons with cleidocranial dysostosis might almost represent a cross section of the general population. Certainly, the vast majority have normal mentality, and in at least 56 cases there has been a direct statement to this effect, such as that the patient was "intelligent." There are, of course, occasional cases in which are manifest a variety

of psychiatric disorders, such as mental deficiency, convulsive disorders, emotional instability and psychoses, and sometimes combinations of these.

Unfortunately, the suggestion of a neurologic component is obscured by the meager number of reports of postmortem studies. Van Neck²⁰ reported a case in which the dura was adherent to the cranium. In Scheuthauer's²¹ case there is said to have been cavities in the frontal lobes the size of pigeon eggs. In 1 of Marie and Sainton's⁴ cases, a man aged 39 had right hemiplegia, and his trouble was diagnosed as syringomyelia. This man died at the age of 52, and Roussy and Ameuille²² confirmed the diagnosis of syringomyelia; they found a large central cavity. In the case of Voisin, de Lépinay and Infroit²³ autopsy was done by Léri and Trétiakoff.²⁴ They observed an inflammatory process in the brain and the meninges and a posthemorrhagic cyst. The most interesting postmortem observation from our point of view is that of Stewart,²⁵ who examined a demented woman aged 47. The frontal poles were small and extremely narrow from side to side. There was poor development of the superior frontal convolutions and of the anterior part of the corpus callosum. There was atrophy or agenesis of the cingular gyrus bilaterally, and section at this level showed absence of mature nerve cells, only a few neuroblasts, almost devoid of processes, being present. The Betz cells of the motor cortex showed chronic degeneration.

Mental deficiency occurs in a few cases of cleidocranial dysostosis. McCurdy and Baer²⁶ reported a case of such deficiency in a Negro aged 52. Still¹⁷ and Heinecke²⁷ have reported cases in which the patients were slow to learn to walk and talk. Kahler²⁸ reported 2

20. van Neck, M.: Autopsie d'un cas de dysostose cléido-crânienne, *Rev. d'orthop.* **21**:323-332, 1934.

21. Scheuthauer, G.: Combination rudimentärer Schlüsselbeine mit Anomalien des Schädels beim erwachsenen Menschen, *Allg. Wien. med. Ztg.* **16**:293-295, 1871.

22. Roussy, G., and Ameuille: Présentation de pièces provenant de l'autopsie d'un cas de dysostose cléido-crânienne héréditaire, *Rev. neurol.* **17**:815-816, 1909.

23. Voisin, R.; de Lépinay, M., and Infroit: Étude clinique et radiographique d'un cas de dysostose cléido-crânienne, *Nouv. iconog. de la Salpêtrière* **20**:227-237, 1907.

24. Léri, A., and Trétiakoff: Autopsie d'une dysostose cléido-crânienne; grosses lésions inflammatoires et hémorragiques méningo-encéphaliques, *Bull. et mém. Soc. méd. d. hôp. de Paris* **47**:1091-1099, 1923.

25. Stewart, R. M.: The Nervous System in Cleidocranial Dysostosis: Report of a Case, *J. Neurol. & Psychopath.* **9**:217-221, 1929.

26. McCurdy, I. J., and Baer, R. W.: Hereditary Cleidocranial Dysostosis, *J. A. M. A.* **81**:9-11 (July 7) 1923.

27. Heinecke, P.: Ueber kongenitale Schlüsselbeindefekte, *Ztschr. f. orthop. Chir.* **21**:553-571, 1908.

28. Kahler, O. H.: Beitrag zur Erbpathologie der Dysostosis cleidocranialis, *Ztschr. f. menschl. Vererb.- u. Konstitutionslehre* **23**:216-234, 1939.

cases of the condition in imbeciles and 7 in feeble-minded persons. Spota and Gonzales²⁹ reported an Argentinian Spaniard with dysostosis and an encephalitic reaction with stupor of narcoleptic type. Lopez Rodriguez³⁰ reported the case of a man aged 27 with cleidocranial dysostosis who was described as oligophrenic. This man had a mental age between 10 and 12 years. It is said that he had great repugnance for every effort, physical or intellectual.

Convulsive disorders are also infrequent. It is probable that some of them may be caused by the increased vulnerability of the brain to injury because of the defective brain case. Molteni³¹ reported the case of a child of 11 years with mental deficiency and epileptiform convulsions. Crouzon and Bouttier³² reported the case of a youth aged 18 who had had frequent convulsions after the age of 16. This was a classic case of epilepsy, with tongue biting, incontinence and loss of consciousness. Massee³³ described the case of a girl aged 15 with dysostosis and an intelligence quotient of 56:

For more than a year she has been subject to epileptic fits which she says are preceded by feeling as though the top of her head were coming off. The attacks occur in varying frequency, sometimes once or twice a day, sometimes once a week.

Stahl³⁵ reported the case of a 4½ year old girl who was an imbecile or a high grade idiot and who was partly deaf and had convulsions. In 1 of the cases of Klinke and Pahlke,³⁴ that of a female infant, tetany was said to have been cured by an antirachitic regimen. A baby reported on by Muldavin³⁵ had convulsions eight days after delivery, but these disappeared in two weeks. In 1 of Marie and Sainton's⁴ cases a brother with dysostosis died in convulsions at the age of 2½ years. Steel and Whitaker³⁶ reported the case of a 24 year old woman who

29. Spota, B. B., and Gonzales, E.: Disostosis cleidocraneana hereditaria y familiar; enfermedad de P. Marie-Sainton, *Rev. Asoc. méd. argent.* **57**:930-933, 1943.

30. Lopez Rodriguez, C.: Contribución al estudio de la disostosis cleidocraneana (con un caso personal), *Actual. méd., Granada* **20**:110-120, 1944.

31. Molteni, M.: Contributo allo studio ed alla casistica della diostosi, cleidocranica, *Arch. di chir. inf.* **1**:345-364, 1934.

32. Crouzon and Bouttier: Sur une forme particulière de la dysostose cléido-crânienne de Pierre Marie et Sainton (forme cléido-crânio-pelvienne), *Bull. et mém. Soc. méd. d. hôp. de Paris* **45**:972-982 and 1099-1100, 1921.

33. Massee, J. C.: Hereditary Cleidocranial Dysostosis: Report of Two Cases, *J. M. A. Georgia* **24**:423-425, 1935.

34. Klinke, K., and Pahlke, H.: Dysostosis cleidocranialis: Bericht über zwei Fälle, *Arch. f. Kinderh.* **91**:46-54, 1930.

35. Muldavin, L. F.: Craniocleidodysostosis in a Boy of Seven, *Brit. M. J.* **2**:13, 1937.

36. Steel, J. P., and Whitaker, P. H.: Case of Cleidocranial Dysostosis, *Brit. J. Radiol.* **10**:613-618, 1937.

had had rare epileptic fits since the age of 17. Russo-Frattasi¹⁴ reported the case of a dysostotic woman who had epileptiform convulsions after scarlet fever. A 15 year old girl with cleidocranial dysostosis reported on by Dowse³⁷ had had epileptic fits from the age of 9 years. She had had uncontrolled gyratory movements of one arm and had lost control of her lower limbs for a short time at the age of 12. Lechelle, Thévenard and Mignot³⁸ described the case of a man aged 29 who fell to his knees three times in one day without loss of consciousness. He had a history of enuresis until the age of 6 and had a para-anal area of anesthesia. Other cases in which enuresis had been reported include those of a 4¾ year old child and a 6½ year old child.

In several reported cases of cleidocranial dysostosis the patients had histories of emotional disturbances. Lyons and Sawyer³⁹ recently recorded a case of a person with psychopathic personality and pathologic emotionality. Abram⁴⁰ mentioned that in his case the patient was intelligent but emotional. Barber and Buchanan's⁴¹ patient and 1 in the family reported on by Villaret and Francoz⁴² were nervous. Thoma and Kalil⁴³ mentioned the "acute sensitivity" of a young woman with dysostosis whom they saw. Boland⁴⁴ reported the case of a youth who made good progress with his studies but had a "roaming disposition." In Hamilton's⁴⁵ case there was difficulty in sleeping. Marchante and Ruiz Cestero⁴⁶ reported the case of a 14 year old boy whose sleep was fitful. The mother of this patient volunteered the comment that the schoolmates made fun of her son and called him "Shorty."

37. Dowse, T. S.: Congenital Deformity of Clavicles, *Tr. Path. Soc. London* **26**:166-168, 1875.

38. Lechelle, P.; Thévenard, A., and Mignot, H.: Dysostose cléido-crânienne avec malformations vertébrales multiples et troubles nerveux: caractère familial des malformations, *Bull. et mém. Soc. méd. d. hôp. de Paris* **52**:1526-1530, 1936.

39. Lyons, C. G., and Sawyer, J. G.: Cleidocranial Dysostosis, *Am. J. Roentgenol.* **51**:215-219, 1944.

40. Abram, J. H.: Cleido-Cranial Dysostosis, *Lancet* **2**:429-431, 1907.

41. Barber, W. W., and Buchanan, L. D.: Congenital Absence of Both Clavicles and Malformation of Cranium (Cleidocranial Dysostosis), *Colorado Med.* **29**:196-201, 1932.

42. Villaret, M., and Francoz, L.: Une famille de quatre sujets atteints de dysostose cléido-crânienne héréditaire, *Nouv. iconog. de la Salpêtrière* **18**:302-343, 1905.

43. Thoma, K. H., and Kalil, F. H.: Clinic of Dental Department of Massachusetts General Hospital and Department of Oral Surgery, Harvard School of Dental Medicine, *Am. J. Orthodontics (Oral Surg. Sect.)* **29**:513-588, 1943.

44. Boland, M.: A Case of Rudimentary Clavicles, *J. A. M. A.* **58**:1442 (May 11) 1912.

45. Hamilton, W. F.: A Case of Congenital Deficiency of Both Clavicles, *Philadelphia M. J.* **4**:720-721, 1899.

46. Marchante, R. F., and Ruiz Cestero, G.: Congenital Cleido-Cranial Dysostosis: Report of Case, *Bol. Asoc. méd. de Puerto Rico* **36**:103-108, 1944.

Besides our case, only 3 cases of the condition associated with a psychosis have been described. Krabbe⁴⁷ described a man aged 28 who gave the impression of having a psychoinfantile personality. He had slightly persecutory ideas and was somewhat deluded. His orientation and mood were satisfactory. Eldridge, Simon and Ramos⁷ redescribed the case of a 55 year old white man which had been previously reported by Cavanagh.⁴⁸ He had suffered from dull headaches and partial deafness for twenty-two years. He had auditory hallucinations, memory defects, impaired judgment, disorientation and unsystematized paranoid delusions. This man talked little, but coherently, and was quiet and seclusive. His Stanford-Binet score was at the 8 year 11 month level, with considerable scatter.

The case in which the postmortem observations were described by Stewart²⁵ had been reported previously by Laverty.⁴⁹ He stated:

The patient's mental condition is one of partial dementia. She recognizes her surroundings, but has no idea of time. She gives her age as 17. Objects such as a book, coin, pencil, are correctly named, and she is able to write her name. She has illusions of mistaken identity, and she states that her mother is in the ward. She makes no attempt to occupy herself, and at times screams loudly for no apparent reason. She feeds, dresses and washes herself, and is in the habit of hoarding rubbish. Probably at one time she had a fair amount of intelligence, but is gradually deteriorating.

This patient had motor weakness and slight spasticity of the lower limbs. She could not stand without assistance, and the strength of the knee jerk was diminished.

In view not only of the rarity and diversity of psychiatric disturbances but also of the dissociation of cleidocranial dysostosis and psychiatric disorders in families, the relationship of the two conditions does not seem an intimate one. In the family we have studied 1 of the dysostotic members has schizophrenia but the other is psychiatrically normal. Similarly, Frets⁵⁰ recorded 2 families in which cleidocranial dysostosis, on the one hand, and convulsions, nervousness, imbecility and insanity, on the other, occurred independently in various members. Rhinehart's¹³ patient, though slow in school, was mentally alert, but a sister without bony anomalies was an idiot and had spastic paralysis. Kahler²⁸ reported that, in addition to 9 cases of mental deficiency among 15 persons with cleidocranial dysostosis, there were also 4 cases

47. Krabbe, K. H.: Dysostosis Cleidocranialis with Metabolic Disturbances, *J. Nerv. & Ment. Dis.* **61**:18-30, 1925.

48. Cavanagh, J. R.: Cleidocranial Dysostosis: Report of Case, *M. Ann. District of Columbia* **3**:11-13, 1934.

49. Laverty, S. J.: Cleidocranial Dysostosis (Anosteoplasia), in *Annual Report of the Metropolitan Asylum Board, London, 1924-1925*, vol. 27, pp. 261-262.

50. Frets, G. P.: Two Cases of Hereditary Dysostosis Cleidocranialis, *Genetica* **12**:513-530, 1930.

of feeble-mindedness and 1 of imbecility among 16 brothers and sisters without dysostosis. He explained this by stating that the earlier generations of dysostotic members were mentally fairly normal but that they had succeeded in finding spouses only among the mentally deficient.

SUMMARY

Cleidocranial dysostosis is a rare developmental disease of the skull, bones of the face, teeth, clavicles, vertebrae, pelvis and phalanges; in fact, it sometimes involves the whole skeleton. The condition ordinarily is inherited as a mendelian dominant. Its incidence is similar in the two sexes and in all races and age groups.

In few of the reported cases were the patients suffering from mental disease. The psychosis in the case here reported manifests connection with the dysostosis only in certain portions of the content and differs in no essential way from the recognized form of schizophrenia. It is evident that the patient's psychologic reactions to his peculiar physical appearance provided the basis for much of the color of the content.

This is in agreement with the impression one gets from a reading of the previously reported cases. In the few persons with mental disease no constant relationship to the dysostosis has been established. The incidence of minor psychiatric disorders in persons with cleidocranial dysostosis is even less frequent than one might have expected considering the social implications of such prominent physical abnormalities.

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ELECTRICAL PARESTHESIAS IN THE EXTREMITIES FOLLOWING INJURY TO THE CENTRAL NERVOUS SYSTEM

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THE condition known as "the electrical sign," or "Lhermitte's symptom," is a rare manifestation of disease of the central nervous system. Patients with the full blown syndrome complain that on bending the head and neck forward paresthesias begin in the base of the neck and radiate into the upper extremities, down the spine and, finally, into the lower extremities. The sensation is variously described as "vibrating," "just like sticking your finger into an electric socket" or "like bumping your funny bone."

In 1918 Babinski and Dubois¹ described the syndrome as a sequela of injury to the neck. One year previously they had reported similar disturbances following head injury. Between 1922 and 1929 Lhermitte and associates² reported that the syndrome was also found in cases of multiple sclerosis. It was Lhermitte's original opinion that it occurred as an early symptom of this disease, but this has been disputed by others. Since Lhermitte's original work, other observers³ have reported a

1. Babinski, J., and Dubois, R.: Pains in the Form of an Electrical Discharge Following Injuries to the Neck, *Presse méd.* **26**:64, 1918; cited by Salmon.^{3f}

2. (a) Lhermitte, J., and Cornil, L.: Heteresthesia Associated with Direct Concussion of the Spinal Cord, *Encéphale* **17**:201, 1922; cited by Salmon.^{3f} (b) Lhermitte, J.; Bollack, and Nicolas, M.: Pains Similar to an Electrical Discharge Following Head Flexion as a Symptom of Multiple Sclerosis, *Rev. neurol.* **31**:56 (July) 1924; cited by Lhermitte.^{2d} (c) Lhermitte, J.; Levy, G., and Nicolas, M.: Electrical Discharge Sensations as an Early Symptom of Multiple Sclerosis, *Presse méd.* **35**:610 (May 14) 1927; cited by Lhermitte.^{2d} (d) Lhermitte, J.: Multiple Sclerosis: The Sensation of an Electrical Discharge as an Early Symptom, *Arch. Neurol. & Psychiat.* **22**:5 (July) 1929.

3. (a) Bériel, L., and Devic, A.: Electrical Discharge Sensations in a Case of Multiple Sclerosis, *Lyon méd.* **141**:559, 1918; cited by Salmon.^{3f} (b) Trioumphoff, A.: Electrical Discharge as a Symptom of Multiple Sclerosis, *Presse méd.* **35**:948 (July 30) 1927; cited by Lhermitte.^{2d} (c) Roger, H.; Reboul-Lachaux, J., and Aymes, G.: Meningeal Dysesthesias Similar to Electrical Discharge on Flexion of the Head as a Symptom of Multiple Sclerosis, *Rev. neurol.* **1**:1052 (June) 1927; cited by Lhermitte.^{2d} (d) Wechsler, I. S.: A Case of Multiple Sclerosis with Unusual Symptoms, *Arch. Neurol. & Psychiat.* **19**:364 (Feb.) 1928; cited by Lhermitte.^{2d} (e) Opalski, A.: Paroxysmal Paralysis (Sensations of Electrical Discharge) in a Case of Multiple Sclerosis, *Rev. neurol.* **1**:281 (March) 1931. (f) Salmon, L. A.: The Sensation of Electric Shock in Multiple Sclerosis, *Bull. Neurol. Inst. New York* **6**:378 (Aug.) 1937.

number of cases of multiple sclerosis in which this symptom appeared. It has also occasionally been reported in cases of tumor or tuberculosis of the cervical portion of the spinal cord and of subacute combined sclerosis of the spinal cord.⁴ However, since the earlier reports of the World War I, few cases of the syndrome occurring after trauma to the head or neck have been recorded.⁵ It is the purpose of this paper to report a series of such cases seen in a large Army neurosurgical and neurologic center.

Triumfov⁶ has recently reported the syndrome in 23 cases of head injury. The wound was occipital in 7 of these cases, parieto-occipital in 5, temporal in 2, temporoparietal in 2 and coronal in 2. The dura mater was involved in 20 cases. He stated the opinion that the intensity probably depends on the proximity of the wound to the foramen magnum and that the syndrome may be the result of injury to the spinal nerve roots or to formation of scar tissue and adhesions in the meninges. He found that the sensation was more severe in the extremity on the side of the injury, that the onset was gradual at the time of healing of the wound (or two or three weeks later) and that the syndrome disappeared gradually in one or two months. In his report, he pointed out that some of the patients experienced a sensation of "weakness" in the distribution of and for the duration of the paresthesia. He found some of his patients considerably disabled by the syndrome, even to the extent that it was necessary for them to hold the head in a fixed position.

REPORT OF CASES

CASE 1.—A private aged 35, on Oct. 14, 1944 sustained a penetrating wound of the right temporofrontal region with a compound, comminuted fracture of the right frontal bone and a lesion of the anterior portion of the second and third frontal convolutions. Foreign bodies were removed from the right frontal lobe. He became ambulatory about Dec. 15, 1944.

At the time of admission to an Army general hospital, on Jan. 22, 1945, he was complaining of occasional headaches and "dizziness" and some pain in the lower part of the back. A few days after admission he began to notice that on bending his head forward he experienced a feeling "like electricity," which began in the low thoracic and upper abdominal regions bilaterally, radiating upward, then into both upper extremities and, finally, out to the fingers. The sensation did not ascend as high as the face, did not appear in the lower extremities and disappeared as soon as he straightened his neck.

4. Hassin, G. B.: Paraplegia in Flexion and the Symptom of Lhermitte, *Arch. Neurol. & Psychiat.* **29**:855 (April) 1933; cited by Salmon.^{3f}

5. Brown, T. G.: A Case of Concussion of Spinal Cord Resultant on a Graze by a Live Shell, with Especial Reference to the Phenomenon of "Heteresthesia," *J. Neurol. & Psychopath.* **1**:54 (May) 1920; cited by Salmon.^{3f}

6. Triumfov, A. V.: The Symptom of "Electrical Discharge" in Brain Injuries, *Am. Rev. Soviet Med.* **2**:350 (April) 1945.

Examination disclosed a right temporal and a frontal cranial defect, hypesthesia of the scalp in the distribution of the right supraorbital nerve, slight reduction in vibratory sensation over the entire left side of the body and reduction in abdominal reflexes on the right side. No cervical roentgenogram was taken.

The patient continued to experience the symptoms of electrical discharge until March 3, 1945, at which time the cranial defects were closed with tantalum plates. After this operation, the sensations disappeared. He was discharged from the hospital on June 25, 1945. A follow-up note from the patient, dated November 21, stated that the paresthesias had not recurred.

CASE 2.—A soldier aged 22 was wounded by shell fragments on Nov. 24, 1944. One fragment caused a compound fracture of the right frontal bone, penetrated the brain and came to rest in the left middle fossa of the skull. Another fragment lacerated the right mastoid region. He suffered immediate left hemiparesis, but this rapidly disappeared. After the wound the patient continued to complain of recurrent generalized headaches. He became ambulatory about Jan. 8, 1945.

About Feb. 15, 1945 he began to note that when he bent his head forward he had a "fluttering sensation," of equal intensity on the two sides, which appeared simultaneously in the upper part of the chest and on the medial sides of the upper and lower extremities. The sensation radiated from the proximal to the distal portions of the extremities, and the patient experienced difficulty in using the extremities during the time that the sensation was present. The sensation was more pronounced after he had held his neck straight for some time. Thus, his symptoms were particularly severe after being in a picture show, when the neck had been immobile for some time. He stated that if he kept his neck bent the sensation finally disappeared, seeming to disappear everywhere at once. He described the sensation as "a kind of an inside shudder, something like an electrical feeling."

The patient was admitted to an Army general hospital on Feb. 14, 1945. Examination disclosed a right frontal cranial defect and a questionable Oppenheim sign bilaterally. The electroencephalogram was normal. No roentgenogram of the cervical portion of the spine was taken.

Cranioplasty was not performed. He was discharged from the hospital August 24.

CASE 3.—On Dec. 2, 1944 a soldier aged 21 sustained a wound from a shrapnel fragment, causing a compound, depressed fracture of the left temporal bone. He immediately lost consciousness and except for two or three short lucid intervals was disoriented for several days. Overseas records indicated that some verbal aphasia was present at first, but this had disappeared by the time of his admission to the Army general hospital, on March 9, 1945. The depressed fracture was elevated, and débridement was performed at a forward installation. He became ambulatory in late December 1944.

About March 1, 1945 he first noted that on bending his head forward he had a "jittery, electric feeling" radiating down the back of his arms and forearms, into the index fingers bilaterally and also into the epigastrium. The sensation appeared to begin spontaneously in the epigastric region, without radiation around from the spine. At no time did he experience any similar sensations in the lower extremities. If he bent his head forward and held it there, the symptoms would disappear within a few seconds. Bending his head forward several times in succession did not appear to decrease the intensity of the symptoms.

Examination disclosed a suggestion of dyspraxia in the right hand, impaired auditory acuity and right homonymous superior quadrantanopsia. A 2 by 3 cm.

defect was present in the left temporal bone. Roentgenograms of the cervical portion of the spine were normal except for slight asymmetry in the atlantoaxial joint, which was probably due to position. The electroencephalogram was abnormal, showing generalized dysrhythmia with focal damage in the left temporo-occipital region.

The patient's symptoms continued unabated throughout March and April 1945, and then they slowly decreased in intensity and frequency and disappeared about June 1, 1945. A tantalum cranioplasty was performed May 24. He was discharged from the hospital June 18, 1945.

CASE 4.—A soldier aged 23 was struck in the right temporal region by a sniper's bullet on June 21, 1945. The bullet passed downward and made its point of exit in the left inframandibular region. Transient paralysis in the upper branches of the right facial nerve occurred. He became ambulatory about July 10.

Ever since sustaining the wound, he had noted mild, generalized headaches, which were relieved by lying down and were gradually improving. About September 1 he noted that when he bent his head forward he had a sensation of shock in his back. This sensation radiated into both upper and lower extremities.

He was admitted to an Army general hospital August 20. Examination revealed nothing abnormal except for a tender scar in the right temporal region. The patient was examined in detail with regard to the electrical paresthesias. By actual timing during the examination, the electrical sensation appeared in the upper extremities one second after bending the head forward and in the lower extremities two seconds later, and it had disappeared completely in about five seconds. The sensation became progressively less intense on bending the head several times in succession. In the upper extremities the sensation radiated into the ends of the fingers, and in the lower extremities it was particularly intense in the calves. No roentgenogram of the cervical portion of the spine was taken.

Cranioplasty was not performed. The patient was discharged from the hospital November 6. A follow-up letter from the patient, dated November 19, stated that he continued to have the electrical sensations, though they were somewhat less marked and that they became more intense after much exercise.

CASE 5.—A private first class aged 20 was wounded by shrapnel fragments on Feb. 9, 1945, sustaining a compound, comminuted fracture of the occipitoparietal region of the skull. He lost consciousness and on regaining consciousness had paralysis of the left upper extremity and complained of scotomas and of hyperesthesia over the entire face and neck. The hyperesthesia and monoplegia disappeared completely in one week, but headaches and postural dizziness persisted until about May 7.

He became ambulatory about April 15; two days later he began to notice that when he bent his head forward he had a sensation "like something running around" in the hypogastric region on each side. Since that time the sensation had spread, and by the time of his admission to the Army general hospital it was described as "like a live wire" and occurred only on his bending the head forward. It would appear in the epigastric region, then radiate to the medial surfaces of the upper extremities and out to the ulnar fingers, simultaneously radiating downward to about the level of the inguinal ligaments bilaterally. The sensation had a duration of three or four seconds. The patient stated that the more rapidly he bent his head the more pronounced was the sensation. If he bent his head slowly, the sensation was not so intense; and after he bent his head several times the sensation would disappear, but would reappear after a period of holding

the neck straight. The symptoms were particularly severe after picture shows, owing to the prolonged immobility of the neck.

Except for a right parieto-occipital cranial defect, the physical and neurologic status was entirely normal on his admission to the Army general hospital on May 19. A roentgenogram of the cervical portion of the spine showed anterior angulation of the entire midcervical part of the spine.

A tantalum cranioplasty was performed on August 8. By August 13 the sensation of electrical shock was less pronounced but had the same distribution. By that time it had also been noted that the sensation could be reproduced by percussion of the spine from about the sixth cervical to the fourth thoracic vertebra. The patient was discharged from the hospital on Sept. 10. A follow-up letter from the patient, dated November 19, stated that the electrical paresthesias had gradually diminished and had been absent since about October 19.

CASE 6.—A soldier aged 38 was struck in the right occipital region by shrapnel fragments on Nov. 27, 1944 and lost consciousness momentarily. On regaining consciousness he noted impaired vision. Examination at forward installations showed no evidence of skull fracture but did disclose left homonymous paracentral scotomas, which persisted.

About April 10, 1945 he began to note that on bending his neck forward he had a sensation "like electricity" radiating into his extremities. This was a distally radiating, "tingling, electric sensation, just like touching a spark plug," which appeared first in the hands and neck and next in the thighs. The sensation did not radiate down the spinal column. If he bent his head forward and held it bent, the sensation would disappear gradually, leaving the lower extremities first. From the time of bending his neck forward to the time of disappearance of the sensation from his lower extremities was about four seconds. If he bent his neck several times in succession, the sensation became progressively less intense; but after a period of holding his neck straight the sensation regained its original intensity. He had no headaches or "dizziness."

The patient was admitted to an Army general hospital March 16, 1945. Examination revealed that the deep reflexes of the left upper extremity were slightly hyperactive. Left homonymous paracentral inferior quadrantic scotomas were present. Slight volitional and emotional weakness was observed in the left lower part of the face. A roentgenogram of the cervical portion of the spine showed congenital fusion of the second and third vertebral bodies but no other abnormality. The electroencephalogram was normal.

The patient was discharged from the hospital on May 28.

CASE 7.—A soldier aged 29 sustained penetrating wounds from shell fragments on Oct. 16, 1944, with a resulting compound, comminuted, depressed fracture of the left occipital bone. The overseas operative note indicated that contusion of the superior longitudinal sinus with possible thrombosis was present. Initially on the left side there were changes in superficial sensation over the face and the upper extremity with mild hemiparesis. The patient had one generalized convulsive seizure on Oct. 20, 1944.

About Dec. 20, 1944 he began to note that on forward bending of the head an "electrical shock sensation" would appear in the back of his neck on the left side and radiate into both upper extremities and then into the trunk and the lower extremity on the left. The patient stated that the sensation was "just like sticking your finger into an electric socket" and that it came only when he bent his head forward.

He was admitted to an Army general hospital Jan. 22, 1945. Examination disclosed hypesthesia and hypalgesia of the left side of the face and of the body above the fourth dorsal segment, with sparing of the seventh cervical segment. The deep reflexes in the left upper extremity were more active than those in the right. There was general contraction of the visual field with paracentral scotomas on the 270 degree meridian bilaterally, mild paresis of the left side of the face of central type and mild deafness in the left ear. No cervical roentgenogram was taken.

The patient was discharged from the hospital on May 12, 1945, after a tantalum cranioplasty, performed March 7.

CASE 8.—A soldier aged 29 was struck by shrapnel fragments on Jan. 2, 1945, sustaining fractures of the left humerus and of the left occipital bone. The humerus healed uneventfully, and the skull fracture was debrided at forward installations. He continued to complain of recurrent headaches and defective vision.

In February 1945, shortly after he became ambulatory, he noted that whenever he moved his head, particularly when he bent it forward, he experienced "shocklike, tingling sensations," generally beginning in the head and radiating through the entire trunk into the lower extremities. The sensation spread "like a wave," never involved the upper extremities and lasted only one or two seconds. It would occur the first time he bent his head but would not recur on successive bending until after he had held the neck straight for a time.

He was admitted to an Army general hospital Jan. 30, 1945. Examination disclosed a right homonymous lower quadrantanopsia and a left occipital cranial defect. An electroencephalogram showed a moderate amount of unlocalized 15 to 20 per cent dysrhythmia. Roentgenograms showed multiple small metallic foreign bodies in the left posterior fossa beneath the cranial defect and anterior angulation at the level of the fourth and fifth cervical vertebrae.

A tantalum cranioplasty was performed on May 4, 1945. He was discharged from the hospital Nov. 17, 1945.

CASE 9.—On April 18, 1945 an officer aged 28 sustained a shrapnel wound, the fragment entering to the left of the midcervical portion of the spine and passing medially to emerge in the suboccipital region in the midline. He sustained a linear fracture of the left parietal region at the same time. He was unconscious for twenty-four hours and on regaining consciousness noticed weakness in the left upper extremity, stiffness and pain in the neck and numbness of the left hand. About June 2, 1945, the feeling of numbness spread to his right hand, and he began to experience a constant tingling sensation in both hands. No objective sensory findings were noted at this time. The weakness in the left upper extremity improved. On April 19 he had a generalized convulsive seizure but has had none since. He became ambulatory in May 1945.

He was admitted to an Army general hospital on June 17, 1945 and shortly thereafter began to notice that when he bent his head forward the tingling sensation in his upper extremities would become exaggerated and would also appear simultaneously in the interscapular region, the spine and the left lower extremity. He described the sensation as "a sort of glow which seems to radiate out," and he stated that it was "similar to the feeling obtained from a shocking machine."

On admission he complained of the paresthesias and of mild, recurrent occipital headaches and postural dizziness. Examination revealed diffuse weakness of the left trapezius muscle and the left arm and forearm (somewhat greater in the muscles innervated by the lower cervical segments). The deep reflexes of the upper and lower extremities on the left were more active than those on the right.

An electroencephalogram, a roentgenogram of the cervical portion of the spine, lumbar puncture and a Pantopaque (an iodized poppyseed oil) cervical myelogram all revealed a normal status.

During his stay in the hospital, the numbness in his upper extremities decreased and was finally noted only in the tips of the fingers on the right and in the entire hand on the left. The headaches disappeared, and the stiffness of the neck improved. He was discharged from the hospital Oct. 20, 1945. A follow-up letter from the patient, dated Nov. 21, 1945, disclosed that the electrical paresthesias had been absent since about Sept. 15, 1945.

CASE 10.—A soldier aged 21 on May 2, 1945 was struck by an enemy rifle bullet, which entered the left lower part of the neck and made its exit through the mouth. As a result of this, he sustained a comminuted fracture of the third cervical vertebra with subluxation of the second cervical vertebra on the third and of the third cervical vertebra on the fourth. Immediately after he sustained this wound, he remembered being able to clench his fists but was unable to move his arms or legs. One-half hour later, while being evacuated, he sustained additional shell fragment wounds in the left arm, the right cheek and the occipital region of the skull, the last not causing fracture. Pain in the shoulder, neck and occipital region on the left side began after this and persisted. About May 10 the tetraplegia began to subside and completely disappeared except for weakness of the left upper extremity in abduction.

He became ambulatory on May 18, 1945. About July 1, he began to notice a "sort of buzzing" sensation throughout his body, which occurred chiefly at night, after he was tired. When he bent his head forward, the buzzing sensation appeared in the back of the neck and radiated into the upper extremities, possibly appearing in the left arm first and being most intense in the fingers. It then appeared in the chest and abdomen but did not radiate into the lower extremities unless he was standing. The sensation spread "like a wave" and was described as similar to an electrical shock. When the patient was seated, it occurred in the upper extremities and the chest; but when he was standing, it appeared in the chest and the lower extremities.

The patient was admitted to an Army general hospital June 23, 1945. Physical examination disclosed some limitation of motion of the neck and, on the left side, weakness of flexion at the elbow, of supination of the forearm and of trapezius function. There was hypesthesia of the left hand generally. The tendon reflexes were more active in the left upper extremity in the right, and Babinski and Chaddock signs were present bilaterally, with an Oppenheim sign on the left. Slight paresis of the left side of the tongue was present, and there was hypalgesia in the distribution of the supraclavicular nerves on the left. Roentgenograms of the cervical portion of the spine taken May 21 showed an abnormal curvature with step formation between the second, third and fourth cervical vertebrae, indicative of dislocation.

The patient was discharged from the hospital Aug. 4, 1945. In a follow-up letter, dated Nov. 19, 1945, the patient stated that the electrical paresthesias had shown great improvement and occurred only when he was unusually fatigued or when he bent the head forward forcibly.

CASE 11.—On March 1, 1945 an officer aged 31 was struck by a shell fragment, which entered the left posterior aspect of the neck and passed to the right and anteriorly, producing an incomplete fracture of the first cervical vertebra and lodging in the tissues of the neck on the right. Immediately after the wound complete sensory and motor loss below the level of the lesion occurred, but

within one-half hour he was able to move all four extremities and sensation returned. He became ambulatory March 20. At the time of admission to an Army general hospital, on June 22, he had improved but continued to complain of pain in the neck and the right shoulder.

Since the first week of May 1945 he had experienced a constant tingling sensation in the tips of the fingers bilaterally. About May 15 he noticed that when he bent his head forward he experienced a sensation like an electrical shock, which began in both elbows and radiated downward into the tips of the fingers. Occasionally, when standing erect, he had the same sensation in the hypogastric region, with occasional radiation into the anterior surfaces of the thighs. The sensation never occurred unless he bent his head forward and then began almost immediately and persisted for about three seconds. There was no tendency for the sensation to "wear out" on successive bendings of the head. The sensation was more intense in the evening than in the morning and tended to become more pronounced after considerable physical exertion.

Examination revealed atrophy of the right posterior cervical muscles and some of the muscles of the right shoulder girdle. These same muscles were paretic. The tendon reflexes in the right extremities were more active than those in the left, and the right upper abdominal reflex was reduced. Roentgenograms of the cervical portion of the spine, taken May 23, showed an old fracture of the right side of the lamina of the first cervical vertebra.

He was discharged from the hospital July 19. A follow-up letter from the patient, dated Nov. 27, stated that the electrical paresthesias disappeared from the lower extremities in August and at the time of the letter were hardly noticeable in the upper extremities.

CASE 12.—On May 7, 1945 a soldier aged 24 was struck in the left infraclavicular region by a bullet, which made its exit in the left suprascapular region. As the result of this wound, there developed left hemothorax and complete paralysis of the left upper extremity, due to an injury of the left brachial plexus. He became ambulatory about May 22. About August 1 he began to note that when he bent his head forward he had electrical sensations radiating down the spine and into the lower extremities. He never experienced this sensation in his upper extremities. He observed that it occurred every time he bent his head forward and did not "wear out" on bending the head several times in succession. It occurred while he was sitting, lying or standing and persisted as long as his head was bent forward.

On admission to the Army general hospital, on June 18, there was complete paralysis of all muscles of the left upper extremity with the exception of those supplied by the eighth cervical and the first thoracic root. On the left side, anesthesia was present over the distribution of the second and third cervical nerves, on the lateral aspect of the shoulder girdle and over the lateral aspect of the arm and forearm. Roentgenograms showed slight anterior angulation at the level of the second and third cervical vertebrae and healing fractures of the posterior portion of the first and second left ribs.

Exploration of the brachial plexus on September 4 showed extensive involvement of the brachial plexus with scar tissue, particularly of the fifth and sixth cervical roots. On the same date a plastic revision of the scar on the left side of the neck was done, and his neck was immobilized for some time. After this operation the electrical paresthesias disappeared. On November 14 the electrical paresthesias were still absent, and he had noted considerable improvement in motor function in the left upper extremity.

CASE 13.—A soldier aged 21, on Nov. 18, 1944 was wounded in the neck by a machine gun bullet, sustaining a compound, comminuted fracture of the fifth, sixth and seventh cervical vertebrae. Immediate paralysis of all four extremities occurred, but all motor function returned in fifteen minutes except in the left upper extremity, where paresis and paresthesia persisted. On November 20 a laminectomy was done. It was found that there was some compression of the spinal cord by the depressed lamina of the seventh cervical vertebrae, and the fractures had obliterated the intervertebral foramen between the sixth and the seventh cervical vertebra on the left. Sensation in his left upper extremity improved for a time after the laminectomy, but about Dec. 20, 1944 he began to notice beginning loss of superficial sensation in the right hand. At the same time he noted that on bending his head forward he had paresthesias "like electricity" radiating down the dorsal surface of his arms and forearms and down the posterior surface of the thighs. There was no radiation into the chest or abdomen. He also observed that on bending his neck forward he had a "pulling" sensation in the ulnar fingers of the left hand.

Examination on March 14, 1945 disclosed generalized weakness of the right upper extremity with paralysis of the extensor carpi ulnaris and probably the opponens pollicis muscle. The radial reflex was more active on the right, and Hoffmann's sign was present on the right side only. Hypesthesia and hypalgesia were noted in the third, fourth and fifth fingers of the right hand, with paresthesia of the volar surface of the thumb and the index finger of the same hand. In the left hand there were paresthesia of the index finger and some paresthesia of the thumb. Otherwise, the neurologic condition was normal. Roentgenograms of the cervical portion of the spine showed a comminuted fracture of the fifth cervical vertebra, multiple small metallic foreign bodies and evidence of a laminectomy.

After admission to an Army general hospital, on Feb. 27, 1945, the patient's electrical paresthesias gradually diminished in frequency and intensity and disappeared completely about April 15, 1945, approximately four months after the onset. He continued to complain of pain and dysesthesia in the index finger of both hands. He was discharged from the hospital July 20, 1945.

CASE 14.—On Nov. 14, 1944 an officer aged 37 was struck in the right supra-clavicular space by a motor shell fragment, which passed down along the ribs and spine, fracturing the fourth and fifth ribs and emerged at this level directly over the spinous process of the fifth thoracic vertebra. An immediate paralysis of all four extremities ensued, with complete loss of sensation below the level of the clavicle. These symptoms disappeared completely in one-half hour. About December 14 he became ambulatory.

On Feb. 2, 1945 he first noted sudden onset of a tingling sensation, "like an electric shock," beginning simultaneously in the inguinal region bilaterally and on the medial surface of the thighs and legs. This occurred whenever he exerted pressure against the scar of exit or whenever he anteflexed his shoulders. The sensation did not "wear out" with repeated production. After a long truck ride he would experience a continuous tingling sensation like electricity in the described areas, which would last about fifteen minutes. Slight weakness in the lower extremities was associated with this tingling sensation.

The patient was admitted to the Army general hospital June 14, 1945. On examination he was normal except for the scar. Roentgenograms of the cervical portion of the spine were not taken but those of the thoracic region showed fracture of the costal processes of the fourth, fifth and sixth thoracic vertebrae on the right.

The electrical paresthesias gradually became less intense and by June 1945 were not present unless the patient became unusually fatigued. He was discharged from the hospital on August 20. A follow-up letter from the patient, dated November 26, stated that the paresthesias were about the same and were present only in his lower extremities. He stated that he did not notice them unless he was very tired or unless he had taken a long, rough ride in some sort of vehicle. Cold weather seemed to aggravate the complaints.

COMMENT

In the cases here reported, the primary wound involved only the head in 8 patients, the head and neck in 2 patients and only the neck in 4 patients.

In patients sustaining some injury to the head, the site of the wound was as follows: frontotemporal, 2; temporal, 2; parietal, 1; occipito-parietal, 1; occipital, 4. This distribution tends to confirm Triumfov's⁶ observation that the syndrome was more frequently associated with posterior than with anterior head wounds.

In the patients who sustained injury to the neck, the site of the lesion was equally distributed in the upper, middle and lower cervical regions.

Of the 10 patients suffering wounds of the head, definite evidence of injury to the brain was present in 8. Of the patients wounded in the neck, evidence of damage to the spinal cord was present in all but 1, and this patient had an injury to the brachial plexus. Four of the patients with wounds of the neck had transient initial tetraplegia.

Analysis of the distribution of the paresthesias indicates that in cases of anterior head wounds the paresthesias are more likely to be limited to the upper extremities and trunk; in cases of posterior head wounds, to the trunk and lower extremities, and in cases of wounds of the neck, to the upper extremities, trunk and lower extremities. It was also noted that in cases of cervical injuries the paresthesias might appear in the neck, upper extremities and trunk while the patient was seated and in the trunk and lower extremities while he was standing. In some instances the sensations did not appear in the trunk at all, while in others they appeared to radiate down the spine.

The average onset of the paresthesias was seventy-five days after the injury, but they appeared as early as thirty-two days and as late as one hundred and thirty-three days. In this series they never appeared until two to sixty-eight days after the patient had become ambulatory (average, forty-one days). After the onset, the duration of the symptoms was variable, sometimes disappearing in a month and sometimes persisting for six months. The disappearance was gradual except in 2 cases, in which symptoms disappeared suddenly after incidental operative procedures.

The electrical paresthesias appear to be the result of injury to the cervical portion of the spinal cord or its membranes, even when the

obvious injury is to the head. This contention is supported by the fact that, in the present series, in 2 of the 10 cases of head wounds there was clinical evidence of concomitant injury to the spinal cord and that, of 6 cases in which roentgenograms of the cervical portion of the spine were taken, evidence of abnormality appeared in 4. The exact pathogenesis of the symptoms is uncertain. Tinel, cited by Salmon,^{3†} expressed the belief that demyelinated sensory neurons might give rise to such symptoms when they were slightly distorted, as in bending the head or neck. This explanation appears to be as satisfactory as any that can be offered at present.

SUMMARY

Pertinent literature on "Lhermitte's symptom" or the "electric sign" is reviewed.

The syndrome is characterized by electrical-like paresthesias radiating into the trunk and extremities on bending the head forward. It is most common with multiple sclerosis and trauma to the head and cervical region of the spine but does occur with other diseases of the spinal cord.

The exact pathologic basis of the condition is unknown, but it appears likely that it results from alterations in the sensory tracts of the cervical portion of the spinal cord. In the cases of trauma to the head and neck reported the paresthesias appeared from a few days to two months after the patient became ambulatory and persisted for one to six months, disappearing gradually.

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HISTOLOGIC STUDY OF THE BRAIN IN EXPERIMENTALLY INDUCED ACIDOSIS

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IN PREVIOUS experiments¹ it was demonstrated that lowering the p_H of the blood led to impairment of excitability of the nervous system to electrical stimulation. The greatest reductions in excitability occurred in reflex arcs. Motor nuclei and peripheral motor nerves underwent the least reductions in excitability in extreme acidosis. In the present experiments an attempt was made to study effects of acidosis on the cytologic structure of the nervous system with methods adequate to reveal slight changes in the structure of nerve cells of the brain.

MATERIAL AND METHODS

Adult guinea pigs of both sexes and various ages were used. In one series of experiments acidosis was brought about by administering a 10 per cent solution of ammonium chloride by stomach tube in doses of 2 to 4 cc. daily over varying periods. In other experiments acidosis was induced by venoclysis with a lactate buffer of p_H 3.4 to 4.0 and by subjection of the animals to atmospheres of 30 per cent carbon dioxide in oxygen. The p_H of the blood was determined with a Coleman p_H meter on samples withdrawn under liquid petrolatum from the heart before the animals were killed. The animals were then anesthetized and fixed, in most instances while still alive, by perfusion with solutions of formaldehyde U. S. P. (3:4) with acacia made isotonic with sodium chloride, according to the method previously described.² The brain was removed and placed in the fixing fluid for four or five days before it was embedded in a solution of pyroxylin of low viscosity, to be sectioned at 40 microns serially. Staining was done with the buffered thionine technic.³ The histologic technic was carefully controlled by staining together sections of an experimental animal and similar sections of a control animal of like sex and weight.

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2. Koenig, H.; Groat, R. A., and Windle, W. F.: A Physiological Approach to Perfusion-Fixation of Tissues with Formalin, *Stain Technol.* **20**:13-22, 1945.

3. Windle, W. F.; Rhines, R., and Rankin, J.: A Nissl Method Using Buffered Solutions of Thionin, *Stain Technol.* **18**:77-86, 1943.

RESULTS

An attempt was first made to study possible effects of chronic acidosis on the structure of the brain. Twelve guinea pigs received daily doses of 10 per cent ammonium chloride by stomach tube for thirteen to thirty days. The p_H of the blood at the end of the experiment varied from 7.32 to 7.08, with a mean of 7.19, in contrast to the p_H of the blood of 9 normal guinea pigs, which varied from 7.55 to 7.30, with a mean of 7.43. The treated animals appeared to be entirely normal throughout the experiment, showing no neurologic signs and maintaining good health. Gross anatomic examination performed with the animal under pentobarbital anesthesia revealed no abnormalities. The brains of 7 of the guinea pigs were studied histologically, a careful comparison being made with sections of the brains of 2 control animals. No alterations in the structure of the brain could be observed in any of these animals. The sections were indistinguishable from the controls. A careful search through all the sections of the brains failed to reveal any intramedullary hemorrhages. Occasionally a few blood corpuscles were observed in the ventricles, but 1 of the control animals showed a similar phenomenon.

Fifteen other guinea pigs were rendered acidotic by forced feeding of a 10 per cent solution of ammonium chloride daily. Eight of these animals were fed the ammonium chloride simultaneously with the 12 in the preceding group. From a few hours to twenty-one days after the feeding of ammonium chloride was begun, in most instances as a result of a slight, and seemingly fortuitous, overdosage of ammonium chloride, the guinea pigs in this series were precipitated into a state of acute respiratory distress, culminating in coma. The p_H values at this time varied from 6.70 to 7.25, with a mean of 7.01. The period of acute respiratory distress lasted from approximately five minutes (in 2 instances) to thirty minutes. Gross examination revealed a state of acute hemorrhagic pulmonary edema in all cases.

Histologic examination of the brains of the 11 animals studied revealed in each instance pathologic changes characteristic of asphyxia. Very small capillary hemorrhages were observed scattered through the brains of 7 of them. In some of these brains the hemorrhages were few and widely scattered. When the sections of the experimental brains were compared grossly with those of the control brains stained simultaneously, it was observed that the sections of 5 brains stained appreciably lighter than the controls. One other brain showed a slight difference. None of the control brains stained more lightly than its accompanying experimental brain. As observed microscopically, small regions around some of the capillaries, especially in the diencephalon, were pale and suggested

perivascular edema. There was no sharp line of demarcation between the area of paling and the more darkly stained tissue. Each of the experimental preparations could be distinguished from its control under the low power lens by the diffuseness of staining. Nerve cells appeared to be less sharply defined than those of the control. Critical examination of the larger neurons of the brain with a high power lens revealed a lack of sharpness in the staining of Nissl bodies. This was most pronounced in the large interneurons, such as those of the red nucleus and the scattered large cells of the reticular formation of the medulla oblongata. In the animals which had suffered the longest period of respiratory distress, many of the large interneurons were noticeably pale, and their Nissl bodies appeared to have undergone some dissolution.

Although a pronounced acidosis existed in this second group of animals, there was no proof that the cytopathologic changes were caused by it. Similar structural changes are seen in cases of asphyxia.⁴ Furthermore, asphyxia occurred with the pulmonary edema in the present experiments. To clarify this relationship, 2 guinea pigs were subjected to rebreathing until they became dyspneic and entered a comatose state. They were perfused while the hearts were beating, and the brains were studied histologically. The brains resembled those of the animals in which pulmonary edema had developed after receiving large doses of ammonium chloride. Structural alterations were of the same order.

Chronic acidosis uncomplicated by pulmonary edema led to no demonstrable changes in structure of the brain. An additional series of experiments was therefore performed in which severe acute acidosis without the pulmonary edema was produced by administration of a lactate buffer of p_H 3.4 to 4.0. This buffer was administered by venoclysis. In each instance a severe dyspnea developed. This appeared to be different from the asphyxial gasping seen in the experiments with ammonium chloride. Respiratory movements were regular, slow to rapid and deep, appearing to involve all the accessory muscles of respiration. The animals were killed thirty minutes to one hour after administration of the buffer. The vascular system was perfused with the fixing fluid, as in the other experiments. At the time the 3 animals were killed the p_H of the blood was found to be 6.0, 6.45 and 6.55, respectively.

The lungs exhibited no edema at autopsy. Two of the 3 animals showed no intracranial hemorrhage; 1 had a small amount of blood around the brain stem and the cervical part of the spinal cord. Histo-

4. Windle, W. F.; Becker, R. F., and Weil, A.: Alterations in Brain Structure After Asphyxiation at Birth, *J. Neuropath. & Exper. Neurol.* **3**:224-238, 1944.

logic study was made of the brains of these 3 animals and of 1 control. No intramedullary hemorrhages were encountered, and no hemorrhages were found on the surface of 2 of the brains. In the other brain, the gross observation of hemorrhage around the brain stem was confirmed, and a considerable quantity of blood was noted in the cerebral aqueduct. There were no other intramedullary hemorrhages.

In spite of the severe acute acidosis produced in these animals and the presence of hemorrhage in the brain of 1, the nerve cells of the brain differed in no way from those in the control specimens. The depth of staining was equal to that of the control, and there appeared to be no dissolution of Nissl bodies anywhere. Thus, the brains of these animals were quite unlike the brains of the guinea pigs which had pulmonary edema after the administration of large doses of ammonium chloride.

Another experiment was performed in which severe acute acidosis was induced by breathing carbon dioxide. A chamber of 75 liter capacity was filled with a mixture of 30 per cent carbon dioxide and 70 per cent oxygen. Two adult male and 2 adult female guinea pigs were used in the experiment. Three normal controls were provided. At the end of five and one-half hours, during which time the gas flowed continuously into the tank, 2 animals were killed. The other 2 animals were killed after three hours in the tank. The p_H of the blood at the time the animals were killed ranged from 6.66 to 7.1, with a mean of 6.94. As in the preceding experiments, the animals were perfused first with an aqueous solution of acacia and sodium chloride (p_H 4.55) and then with the fixing fluid, consisting of solution of formaldehyde U. S. P. (3:4) with acacia and sodium chloride (p_H 4.32). The controls were similarly perfused.

No hemorrhages were observed at autopsy. The brains appeared to be normal in size and appearance. Histologic study revealed no intramedullary hemorrhages. The sections of the control brains stained a little more lightly than the simultaneously stained sections of the experimental brains. The darker staining of the brains of the animals which had been subjected to atmospheres rich in carbon dioxide was due to a more intense staining of the nerve elements themselves rather than to a difference in the staining of the neuroglia. It was not due to any difference in fixation or treatment of the histologic sections. The neurons were not more shrunken but simply took on more of the dye in the case of the experimental animals. The significance of this is not clear. It is a phenomenon not encountered in the other acidotic brains and is perhaps due to the carbon dioxide itself. It was entirely unlike the picture presented by the brains of asphyxiated animals.

COMMENT

The results appear to be in sharp contrast to histopathologic changes in nerve tissue accompanying acidosis reported in the past. Thus, de Crinis⁵ induced acidosis in rabbits and a dog over a period of three days or less by feeding and intravenous injection of sodium biphosphate (10 per cent solution). Among the cytologic changes reported were increase in glial cells, severely damaged ganglion cells and neuronophagia. He also observed these changes in the brains of patients, several of whom died of diabetic coma, several of eclampsia and several of status epilepticus, and he attributed the neurocytologic changes to the acidosis occurring in these conditions. Some of the alterations described in the human brains may well have occurred post mortem. Moreover, acidosis is but one of the disturbances in these clinical entities, and the pathologic changes described could be attributed to another factor. Preterminal asphyxia is capable of producing cytologic changes in the brain, as demonstrated in our present experiment. The changes described by de Crinis in the brains of the animals with experimentally induced acidosis are more difficult to explain. This investigator utilized 1 healthy rabbit as a control. He did not state how soon after death the animal brains were fixed. Thus, it is possible that some of the changes observed were in reality postmortem alterations. In our experiments, the animals were fixed while still biologically alive. Postmortem autolysis and other alterations were thus prevented.

Funagoshi, Choja and Hayami⁶ also reported histopathologic changes in the central nervous system occurring with experimental acidosis. These investigators induced acidosis in rabbits by the subcutaneous injection of hydrochloric acid and phosphoric acid. The cellular changes described included protoplasmic atrophy, pyknosis, atrophy, granulation, coalescence and disappearance of the Nissl bodies, pyknosis, swelling, increase in number, light staining and shrinkage of mitochondria. The mitochondria were not studied in our material; nevertheless, the other neuronal alterations reported by these men were not confirmed by our experiments. Here, too, the time interval between killing the animals and fixation was not reported, nor were control brains said to have been studied.

The absence of visible cytopathologic changes in the brain in experimental acidosis is consistent with the relatively slight effect acidosis is

5. de Crinis, M.: Ueber die Beeinflussung des histologischen Bildes des Zentralnervensystems durch humorale Veränderungen, *Monatschr. f. Psychiat. u. Neurol.* **58**:185-221, 1925.

6. Funagoshi, M.; Choja, N., and Hayami, Y.: Ueber die Veränderung der Mitochondria in den verschiedenen Ganglienzellen bei experimenteller Azidosis und Alkalosis, *Tr. Soc. path. jap.* **27**:459, 1937.

known to have on the electrical excitability of nerve cell bodies and nerve fibers as compared with its effect on spinal reflexes (Koenig and Groat¹). The physicochemical alterations in the central nervous system consequent to acidosis are still but little known and are not necessarily reflected in structural change.

SUMMARY

Guinea pigs were given a solution of ammonium chloride by stomach tube daily over varying periods to induce chronic acidosis. No cytologic changes were observed in the brains. Other animals were made acutely and extremely acidotic by administering a lactate buffer or by subjecting them to atmospheres of 30 per cent carbon dioxide in oxygen. No structural changes could be seen in the nerve cells of the acidotic animals given the buffer solution, although the neurons of the animals breathing carbon dioxide stained appreciably darker than did those of the controls.

When administration of ammonium chloride resulted in coma associated with acute hemorrhagic pulmonary edema, nerve cells of the brain exhibited pathologic alterations which were identifiable as phenomena of asphyxia.

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MENINGOENCEPHALITIS COMPLICATING HERPES ZOSTER OPHTHALMICUS AFTER TREATMENT BY VACCINATION

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SEVERAL investigators have given as explanation of the encephalitis following vaccination and the exanthems the theory of the activation of a latent virus by the vaccination or the exanthem. Van Bastiaanse and co-workers¹ expressed the opinion that the postvaccinal encephalitis occurring in the Netherlands was the result of rousing to active manifestation an encephalitis existing previously in the latent stage. They stated the belief that the virus involved was that of epidemic encephalitis. Netter,² Kraus,³ Demme⁴ and Pette⁵ also favored this idea. The members of the Committee on Vaccination, Great Britain, with one exception, held this opinion; they stated in 1928:⁶ "It is not altogether improbable that vaccination may here and there have precipitated an encephalitis in a person harbouring another virus." Ledingham⁷ wrote:

... Till further notice I take the view that postvaccinal encephalitis is more probably the result of activation of some latent virus parasitising certain hosts and that it is on all fours with the similar syndromes following occasionally measles, chickenpox, etc.

Lust⁸ also maintained that the encephalitis following measles was attributable not to the measles virus but to the awakening of a latent

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2. Netter, A.: *Encéphalites post-vaccinales*, Bull. Acad. de méd., Paris **94**:821, 1925.

3. Kraus, R.: *Zur Aetiologie der postvakzinalen Encephalitis*, Wien. klin. Wchnschr. **40**:185, 1927.

4. Demme, H.: *Welche Schlüsse erlaubt uns das Tierexperiment, auf die Ursachen der Encephalitis nach Kuhpockenimpfung zu ziehen?* Deutsche Ztschr. f. Nervenhe. **105**:177, 1928.

5. Pette, H.: *Das Problem der postvakzinalen Encephalitis*, München. med. Wchnschr. **75**:207, 1928.

6. Report of the Committee on Vaccination, Great Britain, Ministry of Health, London, His Majesty's Stationery Office, 1928.

7. Ledingham, J. C. G.: *Studies on Virus Problems: I. Tissue and Cell Affinities of Viruses and Reactions of the Host*, Bull. Johns Hopkins Hosp. **56**:247, 1935.

8. Lust, F.: *Die paramorbillöse Encephalitis und ihre Folgen*, Monatschr. f. Kinderh. **34**:284, 1926.

neurotropic virus by the measles virus. Schick⁹ stated the opinion that measles lowered the resistance to attack and invasion by a neurotropic virus.

Experimental studies on the activation of one virus by another are few, but Levaditi and Nicolau¹⁰ observed in the rabbit that rabies produced by the injection of the virus of rabies can cause death from neurovaccinia which had been latent. In later experiments¹¹ they claimed that the herpes virus when placed on the nasal mucosa of a rabbit is by the simultaneous inoculation of vaccine on the skin stimulated to produce a fatal herpetic encephalitis. Zurukzoglu¹² inoculated 9 rabbits. Three received 0.5 cc. of herpes virus subcutaneously, followed a few minutes later by 0.2 cc. of smallpox vaccine lymph intravenously. Three rabbits received only the dose of herpes virus subcutaneously, while 3 received only the dose of vaccine intravenously. The 6 control animals remained alive, whereas 2 of the animals which received both viruses died on the ninth and the thirteenth day, respectively, with clinical symptoms of herpetic encephalitis. It is thus suggested that a sublethal dose of herpes virus given subcutaneously was induced to excite encephalitis by the simultaneous injection of smallpox vaccine. Thomsen¹³ found that the simultaneous inoculation into the skin of monkeys of the viruses of vaccine and poliomyelitis may induce the clinical picture of the latter disease, whereas the simple vaccination of the skin with poliomyelitis virus produced no effect of any kind. In the explanation of recurrent fever blisters, Rivers¹⁴ wrote that persons with this condition usually possess an abundance of antibodies against the active agent in their serums.

The paradox has been explained on the basis of the persistence of the virus in the cells of immune individuals who develop crops of blisters whenever subjected to the conditions that are encountered as a result of typhoid vaccination, common colds, exposure to high temperatures for several hours, etc.

In other words, the common cold, which is a virus infection, may activate the virus of herpes febrilis. Fyfe and Fleming¹⁵ reported 9 cases of

9. Schick, in discussion on Lust.⁸

10. Levaditi, C., and Nicolau, S.: Les associations entre ultravirus neurotropes, *Compt. rend. Soc. de biol.* **93**:3, 1925.

11. Levaditi, C., and Nicolau, S.: A propos de l'étiologie de l'encéphalite post-vaccinale, *Compt. rend. Soc. de biol.* **94**:114, 1926.

12. Zurukzoglu, S.: Experimentelle Untersuchungen über Vaccine und Herpes, *Klin. Wchnschr.* **6**:70, 1927.

13. Thomsen, O.: Experimentelle Untersuchungen über die Poliomyelitis, *Ztschr. f. Immunitätsforsch. u. exper. Therap.* **14**:198, 1912.

14. Rivers, T. M.: *Lane Medical Lectures: Viruses and Virus Diseases*, Stanford University, Calif., Stanford University Press, 1939, p. 60.

15. Fyfe, G. M., and Fleming, J. B.: Encephalomyelitis Following Vaccination in Fife, *Brit. M. J.* **2**:671, 1943.

encephalomyelitis following vaccination for smallpox in Fife, Scotland, where 75,326 vaccinations were performed recently, during an epidemic of smallpox. Patients 2, 3, 4 and 6 had acute pharyngitis; patient 7, rales in the chest, and patient 8, a history of recent laryngitis. The findings of these authors suggested, at least, that the hazard of encephalitis during vaccination is much greater when there is concomitant infection in the upper respiratory tract. It is not unreasonable to postulate that several of the infections in these patients were due to viruses.

The following report concerns a case of herpes zoster ophthalmicus in which meningoencephalitis developed subsequent to vaccination employed as treatment for the herpes.

REPORT OF A CASE

History.—A white man aged 60 was admitted to the hospital on Nov. 7, 1945, with coma of several hours' duration. Previous illnesses included pneumonia and bronchitis in 1940 and typhoid in youth. He was married and had 5 children; 1 son was epileptic. He had been vaccinated twice in early life. He was well and working up to Oct. 26, 1945, when he complained of pain in the left frontal area. On the following day his left eye became inflamed. On October 28 a vesicular eruption appeared on the left side of the nose and forehead; the next day the rash had spread. He was vaccinated on October 30, in treatment of the herpes. Except for pain in the frontal region and eye, he was well until November 7. That morning he was awakened with great difficulty and remained awake only long enough to make some unintelligible sounds. Later his extremities became "stiff" and the family noticed occasional movements of the limbs.

Examination.—On his admission, the temperature was 99.4 F.; the blood pressure was 140 millimeters of mercury systolic and 75 millimeters diastolic, and the pulse rate was 80 per minute. There were vesicular lesions, many of them crusted, on the left side of the nose and the left supraorbital region. Two old vaccination scars and a recent vaccination scab was present on the left arm. He was comatose and could not be roused by supraorbital pressure. Tonus in the limbs frequently varied from hypertonicity to flaccidity. At times the lower limbs could not be flexed, even with force. Cogwheel rigidity was elicited at the elbows. There was hyperreflexia in the upper limbs. The Gordon and Mendel-Bechterew signs, a defective plantar response and diminished abdominal reflexes were demonstrated on the left side. Nuchal rigidity was pronounced. The pupils were small; the right pupil reacted to light but not the left. The conjunctiva of the left eye was diffusely reddened and edematous; the cornea was cloudy as a result of posterior precipitates and folds in Descemet's membrane. Fine tremors of the lower jaw were noted from time to time.

Laboratory Data.—The blood count showed 5,500,000 erythrocytes, 100 per cent hemoglobin, and 14,600 leukocytes, with 86 per cent polymorphonuclear leukocytes, 13 per cent lymphocytes and 1 per cent monocytes. The Kahn test of the blood was negative. Urea nitrogen was 16 mg., and the blood sugar 69 mg., per hundred cubic centimeters. The urine was normal. Lumbar puncture on November 7 revealed a clear, colorless fluid with an initial pressure of 120 mm. of water, a cell count of 100 lymphocytes per cubic millimeter, a positive Pandy reaction, a total protein of 120 mg. per hundred cubic centimeters, a colloidal gold curve of 0033220000 and a negative Wassermann reaction. Culture of the spinal fluid disclosed no bacteria.

Course of Illness.—The patient remained in coma for forty-eight hours and then began to respond. On November 9 he was confused as to his age. He thought he was dead and in a morgue and that the examiner was an undertaker. He was extremely suspicious and was slow in responding to questions. There was definite impairment of memory for both recent and remote events. He could not state where he was or give the approximate month or year; he could not name his children. There was no confabulation. The Gordon sign on the left and the nuchal rigidity were still present. The temperature rose on November 8 to 102 F. and fluctuated between 99 and 101 F., until it dropped to normal on November 20. The patient's mental state gradually returned to normal, and on November 27 he was fully alert, cooperative and oriented. There were no delusions or hallucinations. He answered questions briskly, and the family was of the opinion that he was back to his usual state. The cutaneous lesions had practically disappeared by November 23. Lumbar puncture was repeated on November 27. The fluid was clear; the initial pressure was 150 mm. of water; there were 66 lymphocytes per cubic millimeter; the sugar was 52.6 mg., and the total protein 138 mg., per hundred cubic centimeters. Another examination of the spinal fluid was done on December 4 and disclosed clear fluid, with initial pressure of 160 mm. of water, 30 lymphocytes per cubic millimeter and a total protein of 75.5 mg. per hundred cubic centimeters. On December 17 the final spinal puncture showed clear fluid, 50 lymphocytes per cubic millimeter and a total protein of 64 mg. per hundred cubic centimeters.

During his hospitalization the patient received 280,000 units of penicillin and was given 14 Gm. of sulfadiazine between November 7 and November 9. No objective signs of focal involvement of the central or the peripheral nervous system were present on his discharge, on December 30.

COMMENT

The subject of this report had no clinical indications of meningoencephalitis until eight days after vaccination. Lillie¹⁶ suggested lymph vaccination as a treatment for herpes zoster ophthalmicus, and he employed it in 11 cases with success. However, in his first case meningoencephalitis developed ten months after the vaccination. He stated:

... The meningoencephalitis might be of virus origin but I feel that the infection of the skin of the nose was the more probable cause of the condition.

The patient recovered.

Alterations in the cerebrospinal fluid in patients with herpes zoster are found even though no clinical signs of meningoencephalitis are demonstrable. Lymphocytosis is reported in many instances.¹⁷ Achard,

16. Lillie, W. I.: The Treatment of Herpes Zoster Ophthalmicus with Small-pox Vaccine, New York State J. Med. **43**:857, 1943.

17. Chauffard, A., and Froin, G.: Nature évolution et durée de la réaction méningée dans le zona, Bull. et mém. Soc. méd. d. hôp. de Paris **19**:994, 1902. Chauffard, A., and Rendu, H.: Méningite zonateuse tardive dans un cas de zona ophthalmique, *ibid.* **24**:141, 1907. Brissaud and Sicard: Cytologie du liquide céphalo-rachidien au cours du zona thoracique, *ibid.* **18**:260, 1901. Wiegmann, F.: Herpes zoster cephalicus (20 eigene Beobachtungen), München. med. Wchnschr. **81**:1970, 1934.

Loeper and Laubry¹⁸ noted lymphocytosis in 50 per cent of 16 cases. Brown and Dujardin¹⁹ reported lymphocytosis in 28 of 42 cases, and they stated the opinion that the presence of syphilis was not a factor. Schüssler²⁰ found an increase of cells in 16 of 18 cases. Touraine²¹ noted pleocytosis in 50 per cent of 97 cases of localized herpes zoster collected from the literature or occurring in his own experience. In a similar percentage of cases of generalized herpes zoster, the cells were increased.²² Henkel²³ reported an increase of cells in 19 of 30 cases of idiopathic herpes zoster and an elevated protein content in 40.3 per cent of 57 cases. Merritt and Fremont-Smith²⁴ examined the fluid in 8 cases and found an increase in the number of cells in 6 of them. The pleocytosis may persist for a long time,²⁵ even as long as two or three weeks after healing of the herpes.²⁶ Sicard²⁷ reported 2 cases in which the spinal fluid had an increased cellular content ten and thirteen months, respectively, after the appearance of the eruption.

Occasionally a full blown meningoencephalitis develops in a case of herpes zoster. Krumholz and Luhan²⁸ described the autopsy in such a case and briefly summarized 5 others collected from the literature. They mentioned 3 other cases in which no autopsy was performed.

18. Achard, C.; Loeper, M., and Laubry, C.: Le liquide céphalo-rachidien dans le zona, *Bull. et mém. Soc. méd. d. hôp. de Paris* **18**:985, 1901.

19. Brown, W. H., and Dujardin, B.: The Cerebrospinal Fluid in Herpes Zoster, and the Relation of Herpes Zoster to Syphilis, *Brain* **42**:86, 1919.

20. Schüssler, D.: Ueber die Liquorveränderungen beim Zoster, *Dermat. Wehnschr.* **100**:381, 1935.

21. Touraine, A.: Zona et liquide céphalo-rachidien, *Ann. de dermat. et syph.* **6**:289, 1935.

22. Touraine, A.: Le liquide céphalo-rachidien dans les zonas généralisés, *Bull. Soc. franç. de dermat. et syph.* **42**:500, 1935.

23. Henkel, T.: Liquor und Herpes Zoster, Thesis, Köln, 1935; Köln, J. Borowsky, 1936.

24. Merritt, H. H., and Fremont-Smith, F.: The Cerebrospinal Fluid, Philadelphia, W. B. Saunders Company, 1938, p. 137.

25. Touraine, A., and Baumgartner, P.: Zona ophtalmique double, *Bull. Soc. franç. de dermat. et syph.* **41**:736, 1934. Jacquet, P., and Bariety, M.: Zona ophtalmique oculo-sympathique; valeur de l'épreuve des collyres, *Bull. et mém. Soc. méd. d. hôp. de Paris* **50**:1561, 1926. Henkel.²³

26. (a) Georgi, F., and Fischer, O.: Humoralpathologie der Nervenkrankheiten, in Bumke, O., and Foerster, O.: *Handbuch der Neurologie*, Berlin, Julius Springer, 1935, vol. 7, pt. 1. (b) Gordon, I. R. S., and Tucker, S. F.: Lesions of the Central Nervous System in Herpes Zoster, *J. Neurol., Neurosurg. & Psychiat.* **8**:40, 1945. (c) Denny-Brown, D.; Adams, R. D., and Fitzgerald, P. J.: Pathologic Features of Herpes Zoster, *Arch. Neurol. & Psychiat.* **51**:216 (March) 1944.

27. Sicard, J. A.: Le liquide céphalo-rachidien, Paris, Masson & Cie, 1902, p. 182.

28. Krumholz, S., and Luhan, J. A.: Encephalitis Associated with Herpes Zoster: Report of a Case, *Arch. Neurol. & Psychiat.* **53**:59 (Jan.) 1945.

The cellular changes in the spinal fluid associated with postvaccinal encephalitis are much less frequent, and they remain abnormal for a much shorter period. The spinal fluid in the 34 cases of van Bastiaanse¹ was normal except for slight pleocytosis in a few instances. There were no abnormalities in the fluid in the 3 cases of Lucksch.²⁹ Leiner³⁰ reported 3 cases. The highest cell count was 181 cells per cubic millimeter fourteen days after vaccination; six days later it dropped to 10 cells per cubic millimeter. In a second case the cells decreased from 18 to 9 per cubic millimeter in eleven days. Frommel and Baumgartner³¹ found 121 cells per cubic millimeter, with 88 per cent polymorphonuclear leukocytes, twelve and a half days after vaccination in a case of postvaccinal encephalitis. On the following day the cell count had dropped to 71 per cubic millimeter, with 86 per cent polymorphonuclear leukocytes. Rietschel's³² patient had 70 cells per cubic millimeter of spinal fluid fourteen days after vaccination. In Roper's³³ case there were 118 cells per cubic millimeter with 82 per cent lymphocytes, and a total protein of 90 mg. per hundred cubic centimeters fifteen days after vaccination. Two weeks later there were only 14 lymphocytes per cubic millimeter and a protein content of 35 mg. per one hundred cubic centimeters. In the report of the Committee on Vaccination of 1930,³⁴ there were increased white cells in 8 of 19 fatal cases; with 1 exception, the count was below 50.

In the cases of Coyle and Hurst³⁵ and Horder³⁶ no increase in cells or protein was present thirteen and twelve days respectively, after vaccination. Taylor's³⁷ patient had an "excess of leukocytes, not marked." There was no elevation in the globulin. Flexner's^{37a} patient had 13

29. Lucksch, F.: Gibt es beim Menschen eine Vakzine-Encephalitis? *Centralbl. f. Bakt. (Abt. 1)* **96**:309, 1925.

30. Leiner, C.: Ueber zerebrale Krankheitserscheinungen im Verlaufe der Kuhpockenimpfung, *Med. Klin.* **22**:441, 1926.

31. Frommel, E., and Baumgartner, J.: Accidents nerveux consécutifs à la vaccination antivariolique, *Schweiz. med. Wchnschr.* **56**:857, 1926.

32. Rietschel, H.: Ueber Encephalitis postvaccinalis mit Krankendemonstration, *Verhandl. d. phys.-med. Gesellsch.* **56**:99, 1931.

33. Roper, F. A.: Encephalitis Following Vaccination with Recovery, *Brit. M. J.* **2**:103, 1933.

34. Vaccination: Further Report of the Committee, Great Britain, Ministry of Health, London, His Majesty's Stationery Office, 1930.

35. Coyle, C. D., and Hurst, E. W.: Acute Disseminated Encephalomyelitis Following Vaccination, *Lancet* **2**:1246, 1929.

36. Horder, T.: A Case of Cerebral Symptoms Following Vaccination, *Lancet* **1**:1301, 1929.

37. Taylor, J. F.: A Fatal Case of "Post-Vaccinal" Encephalitis, *Lancet* **1**:1302, 1929.

37a. Flexner, S.: Postvaccinal Encephalitis and Allied Conditions, *J. A. M. A.* **94**:305 (Feb. 1) 1930.

cells per cubic millimeter fifteen days after vaccination, and Miller's^{37b} patient had 2 cells five days after vaccination; both patients died. Neal,³⁸ with personal experience of 10 cases, stated that "changes in spinal fluid are usually not marked and normal fluids may be found." Demme³⁹ also stated that in cases of postvaccinal encephalitis the spinal fluid is normal. In Facey's⁴⁰ case encephalitis and a cutaneous eruption developed one month after vaccination. Three examinations of the spinal fluid disclosed no abnormalities.

The patient described in this paper had herpes zoster ophthalmicus, with the probable presence of the herpes virus in the nervous system. As already indicated, in approximately 50 per cent of the cases of herpes zoster there are changes in the spinal fluid without demonstrable clinical signs of meningoencephalitis. The patient was vaccinated as a therapeutic measure, and the second virus apparently activated the latent herpes zoster into full blown meningoencephalitis. It may be argued that the meningoencephalitis was the result of vaccination. There are several points of evidence against this view. Postvaccinal encephalitis follows primary vaccination in the great majority of cases. In the report of the committee on vaccination of 1930,³⁴ 83 of 90 cases occurred in infancy after primary vaccination. In the present case the vaccination was the patient's third. The age of the oldest person reported by the Committee on Vaccinations in 1928 was 55^o; the subject of the present study was 60. The persistence of alterations in the spinal fluid forty days after the initial puncture is also in favor of a herpes infection. Whether this patient would have had meningoencephalitis as a complication of the herpes zoster if he had not been vaccinated is difficult to say. In the majority of cases such a complication appears a little later in the course of the illness. However, one cannot ignore the probable role of the vaccination as a precipitating factor. Localized herpes zoster following vaccination has been noted.⁴¹ Unfortunately, I was not able to conduct animal experiments with the spinal fluid. Even if the animals inoculated were to show histologic changes, it would not necessarily prove that vaccination produced the clinical picture in the patient, since the virus of vaccinia is occasionally present in the blood, in various organs and even in the spinal fluid in cases of severe reactions to vaccination without

37b. Miller, M. K.: Four Types of Encephalitis, *J. A. M. A.* **97**:161 (July 18) 1931.

38. Neal, J. B.: Encephalitis, New York, Grune & Stratton, Inc., 1942, p. 104.

39. Demme, H.: Liquorbefunde bei akuten Infektionen des Nervensystems, *Zentralbl. f. d. ges. Neurol. u. Psychiat.* **54**:133, 1930.

40. Facey, R. V.: Encephalitis with Skin Eruption After Vaccination, *Lancet* **2**:669, 1942.

41. Dumont, J.: Vaccine et zona, *Bull. et mém. Soc. méd. d. hôp. de Paris* **46**: 1036, 1922. Frommel and Baumgartner.³¹

complications.⁴² In addition, several investigators⁴³ found that the virus of vaccinal encephalitis, as well as the herpes zoster virus, could not be transmitted to animals.

SUMMARY

An instance of meningoencephalitis with recovery in a 60 year old patient with herpes zoster ophthalmicus is reported. The onset of the nervous complications appeared eight days after the patient was vaccinated in treatment for the herpes. It would seem that the herpes virus was activated into a full blown meningoencephalitis by the vaccinia virus. The persistence for weeks after the onset of changes in the spinal fluid occurring with meningoencephalitis complicating herpes zoster is confirmed in this case.

1882 Grand Concourse (57).

42. Herzberg-Kremmer, H., and Herzberg, K.: Untersuchungen über post-vakzinale Enzephalitis, *Zentralbl. f. Bakt. (Abt. 1)* **119**:175, 1930.

43. Marinesco, G.: Contribution à la pathogénie et à la physiologie pathologique du zona zoster, *Bull. Acad. de méd., Paris* **88**:487, 1922. Blanc, G., and Caminopetros, J.: Contribution à l'étude expérimentale de zona, *Bull. Soc. franç. de dermat. et syph.* **29**:294, 1922. Marinesco, G., and Draganescu, S.: Contribution à la pathogénie et à la physiologie pathologique du zona zoster, *Rev. neurol.* **39**:30, 1923. Doerr, R.: Ergebnisse der neueren experimentellen Forschungen über die Aetiologie der Herpes simplex und des Zoster, *Zentralbl. f. Haut- u. Geschlechtskr.* **16**:481, 1925. Cole, R., and Kuttner, A. G.: The Problem of the Etiology of Herpes Zoster, *J. Exper. Med.* **42**:799, 1925. Gordon, M. H., cited in the Report of the Committee on Vaccination.⁶ Esser, A.: Die Hirnschädigungen nach Pockenschutzimpfung, *Virchows Arch. f. path. Anat.* **278**:200, 1930. van Bastiaanse and others.¹ Kraus.³ Denny-Brown, Adams and Fitzgerald.^{26c}

SURGICAL TREATMENT OF SYRINGOBULBIA AND SYRINGOPONTIA

Report of Two Cases

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SURGICAL operation on the brain stem for the relief of syringobulbia and syringopontia is herewith reported in 2 cases, with successful outcome. In a search of the literature I was unable to find reports of or references to precedence in the direct surgical attack on this entity. Its counterpart in the spinal cord, syringomyelia, has been successfully treated and reported on by a number of authors.

Frequently called the Puussepp procedure, the operation for syringomyelia was performed by Elsberg¹ at an earlier date, and Abbe and Coley were credited by Adelstein² with the first report of this procedure, in 1892. Adelstein, Frazier,³ Putnam,⁴ Kuhlendahl,⁵ Cox,⁶ Worster-Drought and associates,⁷ Woods and Pimenta⁸ and others cited by them have all reported on the surgical treatment of syringomyelia. Whereas Frazier,⁹ in evaluating 2 of his own cases and 14 early cases collected from the literature, found notable improvement in 50 per cent, Woods and Pimenta,⁸ in 1944, in an analysis of the 20 cases which they reported, averred that the surgical treatment of syringomyelia, with or without roentgen therapy, has not produced the hopeful results previously reported in the literature. It should be emphasized, as several of these

1. Elsberg, C. A.: Surgery of Intramedullary Affections of the Spinal Cord: Anatomic Basis and Technic, J. A. M. A. **59**:1532-1536 (Oct. 26) 1912.

2. Adelstein, L. J.: The Surgical Treatment of Syringomyelia, Am. J. Surg. **40**:384-395 (May) 1938.

3. Frazier, C. H.: Drainage of a Syringomyelic Cavity, Twice in the Same Patient, Three Years Intervening, J. A. M. A. **101**:1228 (Oct. 14) 1933.

4. Putnam, T. J.: Syringomyelia: Diagnosis and Treatment, M. Clin. North America **19**:1571-1582 (March) 1936.

5. Kuhlendahl, H.: Die operative Beinflussbarkeit der Hydromyelia und Syringomyelia, Deutsche Ztschr. f. Nervenhe. **140**:1-27 (Feb. 22) 1936.

6. Cox, L. B.: On the Origin and Treatment of Syringomyelic Cavities, M. J. Australia **1**:481-483 (March 12) 1938.

7. Worster-Drought, C.; Wakeley, C. O. G., and Shafar, J.: The Surgical Treatment of Syringomyelia, Brit. J. Surg. **29**:56-73 (July) 1941.

8. Woods, W. W., and Pimenta, A. M.: Intramedullary Lesions of the Spinal Cord: Study of Sixty-Eight Consecutive Cases, Arch. Neurol. & Psychiat. **52**:383-399 (Nov.) 1944.

9. Frazier, C. H., and Rowe, S.: The Surgical Treatment of Syringomyelia, Ann. Surg. **103**:481-497 (April) 1936.

authors have already done, that the indications for operation should be those already well established for operation on the central nervous system. In the case of syringomyelia the most important indication is a relentless progression of symptoms, coupled with evidences of abnormal tension and of cerebrospinal fluid block and correct localization. If these criteria are applicable to syringobulbia, the operation may be urged. Certain reservations, based on the disseminated and progressive nature of the basic pathologic state, must be borne in mind. It must be realized that many of the symptoms will, of necessity, remain static because of the essentially destructive nature of the disease. However, the signs and symptoms of increasing pressure will be relieved, and if the end stage of cavity formation has been reached at the time of operation the further progress of the disease may be halted. Furthermore, not all the paralytic symptoms are necessarily due to irreversible changes in the cellular groups and neural pathways involved. Some may be produced merely by direct hydrostatic pressure from the adjacent cavity and after relief of this tension may show improvement.

REPORT OF CASES

CASE 1.¹⁰—A 35 year old timekeeper and clerk was treated in the Neurological Institute of New York from July 17 until Sept. 16, 1941, with craniectomy in the posterior fossa on Aug. 21, 1941. He entered the hospital with the following chief complaints: (1) Blurred vision, later becoming true diplopia, of sudden onset, in the summer of 1939, while he was reaching to catch a ball during a game. The disturbance recurred intermittently, chiefly on movements of the head, and had a progressive course. (2) Awkwardness of the left hand, first noted in typewriting in the late summer of 1939, and later, in more pronounced form, in piano playing, in the autumn of 1939. (3) Attacks of light headedness and sensations of being suspended in air, accompanied with diplopia and precipitated by looking to the left; onset in the winter of 1939. (4) Staggering gait and tendency to drag the left foot; onset in the spring of 1941. (5) Tinnitus, bilateral, occurring spontaneously at times but chiefly on turning the head sharply; onset in the spring of 1941. (6) Coarse tremor of the left forearm, of pronation-supination type, occurring frequently, on attempting purposive movements; onset in the summer of 1941. (7) Nuchal pain and headache, radiating from the base of the skull, posteriorly, up over the vertex in the midline. This occurred chiefly on flexion of the head, as when he was lacing his shoes; it also was noted on extension of the head and on coughing, sneezing and straining at stool. Onset was in the summer of 1941. (8) Nausea and vomiting, occasional, sometimes projectile, preceding his admission to the hospital.

The past history was not remarkable except for a chronic cough of twelve years' duration, diagnosed as bronchiectasis and productive of a few teaspoons of pus daily, which was not foul tasting or malodorous. He also had had two head traumas. The first had occurred twenty years before and was associated with retrograde amnesia but not unconsciousness; the second, occurring after the onset of his present illness, the result of tripping on the stairs, was associated with

10. Case 1 is from the Department of Neurology, Columbia University College of Physicians and Surgeons, and the Neurological Institute of New York.

bleeding from the ear, ecchymosis about the eyes and a state of being dazed, without unconsciousness.

General Physical Examination.—The patient was slender and prematurely gray; he did not appear acutely ill on admission but grew progressively worse during the period of preoperative observation in the neurologic and neurosurgical services. The general physical condition was normal.

Neurologic Examination.—Gait: He walked with a broad base and showed a tendency to stagger in either direction and to veer toward the right. No hemiplegic attitude was obvious; but the associated swing of the left arm in walking was diminished, and in crawling he tended to slide the left hand forward instead of lifting it, as he did the right.

Motor Status: Motor power was reduced in both left extremities, yet he was able to walk on both his heels and his toes. Equilibration was normal. Non-equilibratory coordination tests revealed a tremor of the left forearm in both pronation and supination; this occurred intermittently when the patient was at rest or was leaning on his palm. Succession movements were poorly performed in both left extremities; there was past pointing in the left hand, and check and rebound phenomena were abnormal in the left arm. Posture holding was normal.

Reflexes: The tendon reflexes were hyperactive throughout but were more exaggerated on the left. The superficial reflexes were all present and equal, except that the plantar response on the right was not as definite as that on the left.

Cranial Nerves.—First: The olfactory nerves were normal.

Second: Visual acuity and the visual fields were normal on gross testing. The vessels were normal, but the edges of the disks were blurred on the nasal side.

Third, Fourth and Sixth: There was pronounced, sustained nystagmus on looking toward the left, and some on lateral gaze to the right. There was slight ptosis of the left upper eyelid. The right pupil was 5 mm. larger than the left.

Fifth: Sensation was normal over the face. Some equivocal sensory alteration was noted on the anterior portion of the left side of the tongue. The corneal reflex was decreased in strength on the left side, the loss developing during the period of observation. The masseter and temporal muscles were paralyzed on the left side.

Seventh: There was weakness in all the muscles of expression on the left side of the face. The greatest weakness was in the lower portion of the face, simulating facial paresis of central origin except that spontaneous blinking was also affected.

Eighth: Hearing was normal except for audiometric diminution in the upper tones in both ears. Responses to the caloric test were not determined.

Ninth and Tenth: There was no gross loss of function, but the pharyngeal and palatal reflexes lagged on the left side.

Eleventh: No weakness was demonstrable in the sternocleidomastoid or the trapezius muscles; yet the patient held the head rotated toward the left, with the chin elevated.

Twelfth: The tongue deviated slightly toward the left.

Mental State.—The mental condition of the patient was normal.

Special Studies.—Roentgenographic examination of the chest revealed nothing abnormal. Roentgenograms of the skull revealed no evidences of increased intracranial pressure and no abnormalities of the sphenoid or petrous ridges or of the internal acoustic meatuses.

Electroencephalographic studies revealed no abnormalities of focal nature, but there was a diffuse mild abnormality. Phase reversal was noted in both frontal regions, and there was low voltage, fast and slow activity throughout.

Pneumoencephalographic examination was performed on Aug. 17, 1941, 80 cc. of fluid being replaced with 75 cc. of air. The lateral and third ventricles were moderately dilated, the cisterna pontis was narrowed, and the anterior extension of the left cisterna magna was narrowed, while only the posterior portion of the cistern on the right was visualized. The roentgenographic diagnostic impression was that of tumor in the posterior fossa, in the cerebellum rather than in the brain stem, and to the right of the midline.

Routine laboratory tests of the blood and urine revealed no abnormality. The protein of the cerebrospinal fluid measured 60 mg. per hundred cubic centimeters.

The preoperative clinical diagnosis was tumor of the brain stem, probably glioma, with an extra-axial growth, possibly meningioma, a less likely probability. Because of the relentless progression of his symptoms and beginning deterioration of his general status, operation was advised.

Operation.—On August 21, with the use of local anesthesia and with the patient in the sitting position, a craniectomy was carried out in the posterior fossa. On opening the dura, the cerebellar tonsils were noted to be herniated into the spinal canal to a considerable extent. Exploration was first carried out in the left cerebellopontile angle, where, at the point of emergence of the trigeminal nerve, an abnormal protuberance of the brain stem was encountered. Its thin wall was opened by blunt dissection, and an unmeasured quantity of yellow fluid escaped. No tumor tissue was observed. The depth of the remaining cavity was about 1 cm. The interior of the fourth ventricle was then explored, and the left half of the floor of the ventricle was observed to be tumefied and greatly deformed. Just above the acoustic striae there was an obviously thin-walled cyst, which was entered by blunt dissection and evacuated of its yellow fluid contents. The size of the cavity was not accurately determined. A specimen of the wall was removed for histologic examination. The maneuvers described had no appreciable effect on the patient's vital condition or immediate symptoms.

Pathologic Report on Surgical Specimen (Dr. Abner Wolf).—Gross Examination: The specimen consisted of two small pieces of tissue, measuring 7 by 5 by 4 mm. and 4 by 5 by 6 mm. respectively, which had the consistency of soft rubber. The tissue was pale gray, and the outer surface was smooth and uniform in appearance.

Microscopic Study: The specimen consisted of a number of fragments of neural tissue. Some of these contained clusters of nerve cells and tracts the architectural arrangement of which suggested that of the normal brain stem. Others contained no nerve cells or only a few sclerotic cells. In these areas there appeared to be an increase in glial fibers, and there were homogeneous, twisted fragments of deeply-staining reddish material, the so-called Rosenthal fibers. Occasional perivascular lymphocytic infiltrations were seen in such zones. On the surface of some of these areas were what appeared to be flattened ependymal cells. This tissue at times appeared to be rather edematous. No tumor tissue was noted in the specimen. The dense mass of glial tissue, the Rosenthal fibers and the adjacent, well preserved parenchyma of the brain stem suggested that the specimen was from the wall of a syrinx.

The pathologic diagnosis was syringopontia. There was no gross or microscopic evidence of tumor (fig. 1).

Postoperative Course.—The patient's complaints referable to increased intracranial pressure were relieved by the operation, and he made an uneventful recovery from the procedure. Postoperatively, there was a coarser and more frequent tremor of the left arm and no appreciable change in the symptoms of cranial nerve palsy,

and the ataxia remained. Six months after operation, on a follow-up visit to the clinic the patient was ambulatory but was still very ataxic. He was last observed, for the purpose of this report, fourteen months after operation, at which time he was not yet employed but was seeking work consistent with his status, to which he was becoming accustomed. He still retained, however, the neurologic deficits which he had before operation.

CASE 2.—The 35 year old widow of a deceased Army officer entered an Army general hospital on Aug. 8, 1943, with the following chief complaint: At intervals for many years she had had a peculiar, shocklike sensation referable to the atlanto-occipital region. This had occurred particularly on movement of the head and was a distressing sensation. The same movement precipitated a similar shocklike sensation in the lower extremities. Until about June 1943 she had had these sensations intermittently, during self-limited periods. However, for the several

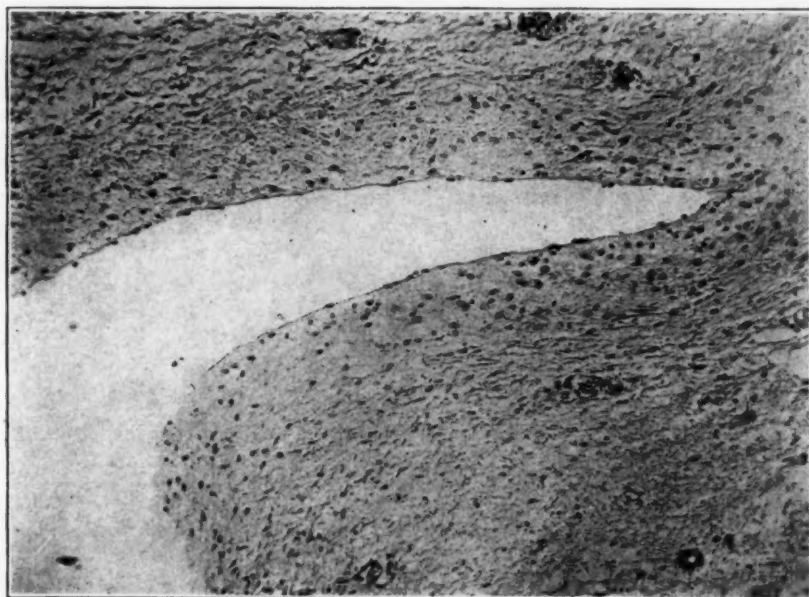


Fig. 1 (case 1).—Section from the wall of the cavity in a case of syringomyelia.

weeks before her admission to the hospital they had become severe and persistent and had been associated with the following complaints: (1) pain in both upper extremities, chiefly on the right; (2) subjective numbness on the right side of the face and tongue; (3) a subjective sensation of weakness in the throat, making breathing difficult; (4) a disturbance in salivation, which she referred to the right side, under the tongue and described as "tangy" or "cankery"; (5) a tingling sensation on the right side of the tongue; (6) nausea and vomiting, and (8) blurred vision.

Neurologic Examination.—Gait: The gait was moderately ataxic, and she was unsteady in standing on either foot alone or on both feet together with the eyes open or closed.

Motor Status: There was no gross loss of motor function.

Reflexes: There was moderate hyperreflexia over the entire right side.

Sensation: There was slight diminution of sensation to all modalities over the right side of the forehead and the bridge of the nose. There was also some sensory diminution on the right half of the face and tongue, on the right posterior wall of the pharynx and of the palate and on the left side of the trunk and the left extremities.

Cranial Nerves.—First: No subjective or objective loss of olfactory sense was apparent.

Second: There was no definite papilledema, but the veins were dilated in both fundi, particularly in the upper portion of the right and the lower portion of the left.

Third, Fourth and Sixth: There was subjective blurring of vision. No weakness of the ocular muscles was apparent, but a fine, rhythmic nystagmus appeared on lateral gaze to either side and on looking upward.

Fifth: The motor portion of the nerve was normal; sensation in the distribution of this nerve has been described in the preceding section.

Seventh: The seventh nerve was normal.

Eighth: Hearing was 20/20 bilaterally. The caloric test with injection of ice water into each ear produced nystagmus in twenty seconds of one hundred and fifty seconds' duration. This response was interpreted by the otologist as showing a hyperirritable labyrinth.

Ninth and Tenth: There were subjective disturbance of salivation, as noted previously, and diminished sensation over the pharynx and palate. Speech was abnormal, being "palatal," but there was no hoarseness or paralysis of the palate, and the uvula moved in the midline.

Eleventh: The nerve was normal.

Twelfth: There were slight, but definite, atrophy of the lateral border of the right side of the tongue and deviation of the tongue toward the right on extension.

Laboratory Data.—The blood and urine were normal. Examination of the spinal fluid showed no trace of globulin, but the total protein was increased to 52 mg. per hundred cubic centimeters. There were 2 white cells per cubic millimeter; these were lymphocytes. The colloidal gold curve was 1111100000.

Preoperative Diagnosis.—The diagnostic probabilities were: (1) tumor, (2) syringobulbia and (3) adhesive arachnoiditis.

Clinical Course.—Under observation, the patient grew progressively worse, complaining of nausea, vomiting and headaches, which were nuchal and occipital. Her condition began to become alarming, and inasmuch as the neurologic picture was patent in its localization of the lesion in the medulla, an exploratory operation was decided on.

Operation.—On October 1, with the patient under ether anesthesia, an upper cervical laminectomy and a suboccipital craniectomy on the right side, extending across the midline, were performed. When the dura was opened, a large cyst was disclosed, occupying the upper segment of the cervical part of the cord and the medulla and filling the entire space usually occupied by the cisterna magna and the inferior portion of the fourth ventricle. This cyst was overlaid with a somewhat thickened and vascularized arachnoid membrane. At the lateral extent of the cisterna cerebellomedullaris there was a small cistern containing clear fluid. The dura was held in retraction by silk sutures, and the cystic mass was then explored. It was observed to extend from the spinal cord opposite the junction of the first and second cervical laminae into the fourth ventricle. It had displaced both cerebellar hemispheres upward and laterally, hollowing out a large space

between them. There were small blood vessels coursing over the surface of this cystic mass. It was incised in an avascular area and approximately 30 cc. of yellow cystic fluid aspirated. Some of this was sent to the laboratory for examination. Within this cystic cavity, the roof of which was opened widely with a longitudinal incision, no tumor tissue could be seen. The walls were of a yellowish appearance. At the inferior portion of the cyst, where it joined the body of the medulla, there were several large veins. With the deflation of the cyst it was seen that the upper portion, which filled the fourth ventricle, was merely the wall. The lower portion, at the junction of the medulla and the spinal cord, was solid. This portion was rather vascular. A small section of the wall of the cyst was removed for examination. After evacuation of the cyst, the dura was closed with

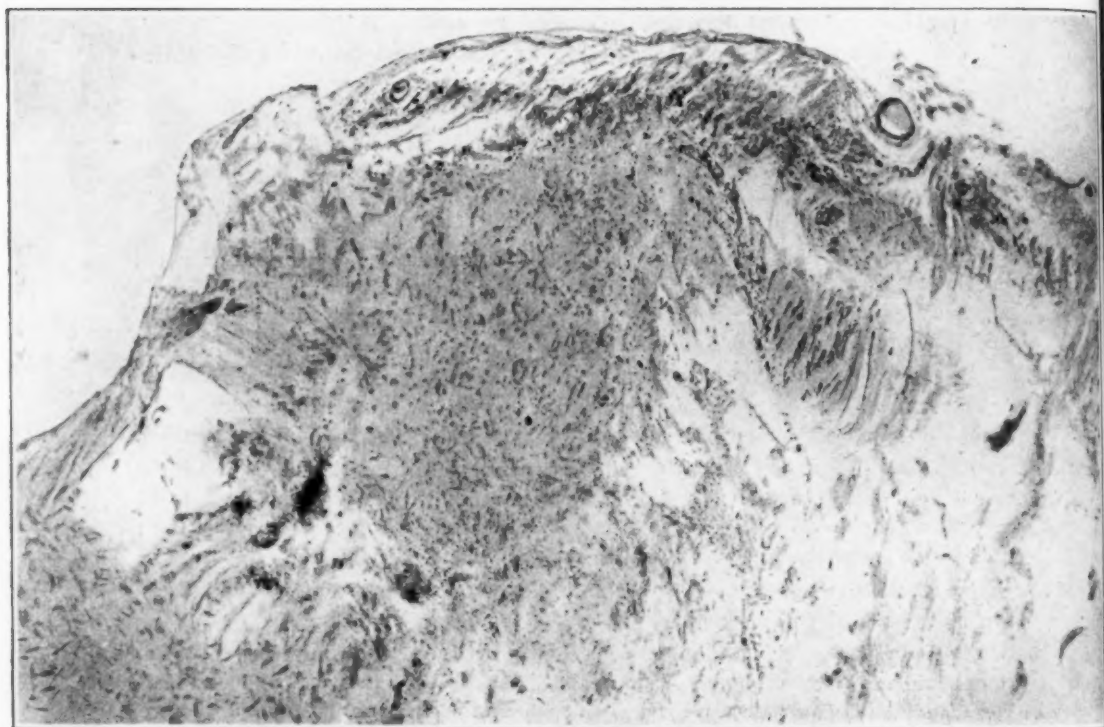


Fig. 2 (case 2).—Degenerative nerve tissue removed at operation in a case of syringobulbia; magnification about 57.

a lock stitch of silk, and the muscles, fascia and skin were closed with interrupted black silk sutures. The condition of the patient remained excellent throughout the operation, but she received two transfusions of blood, as well as dextrose, during the operation as supportive measures.

Diagnosis.—The postoperative diagnosis was syringobulbia.

Postoperative Course.—The patient made an uneventful recovery. On the sixth postoperative day the sutures were removed, and the wound healed by primary intention. The patient was able to get up and about gradually; when she was reexamined on Nov. 24, 1943, she was free from headaches but had some subjective dizziness and epigastric distress, with slight nausea and occasional vomiting. She

had slight ataxia in walking but no nystagmus or past pointing. The hyperreflexia remained over the entire right side, and there was slight diminution of sensation over the right side of the forehead and the bridge of the nose, as well as over the left upper extremity. The optic disks and vessels appeared normal. In summary, the symptoms referable to increased intracranial pressure, and to some degree the involvement of the cranial nerves, had regressed satisfactorily at this time. The loss or alteration of function in the long tracts remained about the same. She was discharged from the hospital on Dec. 7, 1943.

The patient made a very satisfactory recovery, and after a rest of six months she was able to earn her livelihood as a clerk. When last examined, about eighteen

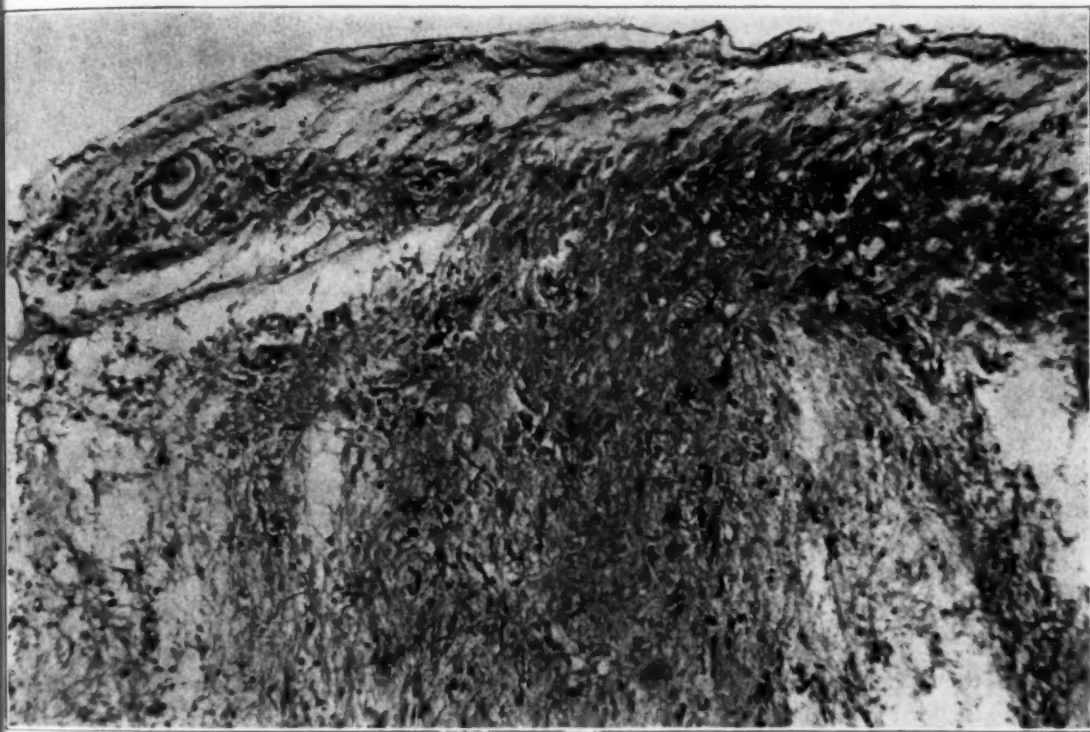


Fig. 3 (case 2).—Tissue similar to that shown in figure 2; magnification about 120.

months after operation, she no longer had atrophy of the tongue, and the other signs previously enumerated were minimal. However, she had referred pain about the breast, and the possibility of developing syringomyelia must be entertained.

Comment on Cases.—"Syringopontobulbia" is the proper designation for the condition in case 1, on the basis of the anatomic location of the lesions and the clinical syndrome. How much part an extension into the midbrain itself may have played in the causation of the pupillary inequality, the slight ptosis of the left lid and the very coarse, alternating tremor of the left arm cannot be determined but is of speculative

interest. The differential involvement of the trigeminal nerve is of note, since the pathways for pain and temperature sense, being bulbo-spinal, were barely affected, whereas involvement of the motor nucleus, being pontile, caused complete paralysis. The subjective sensation of levitation produced by certain head movements is the most interesting symptom. I do not recall that this sensation of being suspended in air has been expressed by any other patient with a cerebellar or cerebello-pontile lesion. From the neurophysiologic standpoint, it is difficult to localize, except that it is generically of vestibular pathway origin. But whether in this case it was an aberration of transmission of normally engendered stimuli within the semicircular canals or was precipitated by impact of fluid in the syrinx against the intra-axial pathways is, again, a matter for speculation. The peculiar tilt of the head, simulating that seen with paralysis of the sternocleidomastoid muscle, was doubtless of vestibular pathway origin. The particular type of facial paralysis was somewhat unusual and worthy of note. Whereas the lower portions of the face were severely affected, voluntary function in the upper portions was, by contrast, well preserved. Yet, as seen on simple observation of the patient's face at rest, the restriction of automatic blinking of the left eye was the signal evidence of a lesion of the lower motor neuron type. Inasmuch as the lesion was pontile and the sixth nerve was not affected, I attribute the facial paralysis to involvement of the nucleus, rather than of the pathway or the peripheral course of the nerve. The occurrence of syringomyelic states with the Arnold-Chiari syndrome makes the observation that the cerebellar tonsils were herniated into the spinal canal of some importance. At operation I accepted this herniation as typical of that observed during operations for tumor of the posterior fossa. However, the general symptoms of pressure had occurred late in the illness, and the patient had no papilledema. Also, the exploration into the cerebellopontile angle was easily made, as is not the case with tumor. Inasmuch as the spinal canal proper was not inspected, it cannot be known whether the elongated tonsils were attached to and drawn down by the spinal cord or whether they were free. That the fourth ventricle and the medulla were in their normal position, and not drawn down, is certain. These facts are emphasized because of reports in the literature of the association of other fragments of status dysraphicus and myelodysplasia with these syringomyelia-like conditions, as discussed by Lichtenstein¹¹ and Nelson.¹² Such syndromes are the Arnold-Chiari malformation and other syndromes of anomalous

11. Lichtenstein, B. W.: Distant Neuroanatomic Complications of Spina Bifida (Spinal Dysraphism): Hydrocephalus, Arnold-Chiari Deformity, Stenosis of the Aqueduct of Sylvius, etc.; Pathogenesis and Pathology, *Arch. Neurol. & Psychiat.* **47**:195-214 (Feb.) 1942.

12. Nelson, J.: Intramedullary Cavitation Resulting from Adhesive Arachnoiditis, *Arch. Neurol. & Psychiat.* **50**:1-7 (July) 1943.

segmental development, usually with defects or abnormalities of the skull and/or spine demonstrable in roentgenograms. In the present cases there were no bony abnormalities. No indications that the cystic degeneration was secondary to an extra-axial cause were noted.

Case 2 represents a syringobulbomyelia and exhibits various features of a lesion in this particular location, simulating the well known syndrome of the posterior inferior cerebellar artery, in the domain of which it occurred. In contrast to the picture in case 1 are the findings referable to the trigeminus in the second case. Whereas in the first case there was complete motor paralysis and little sensory change, in the second case there was of necessity involvement of the descending tract only, and therefore no motor loss, but subjective and objective changes on the forehead and face. Of greater surgical interest, however, is the regression of the atrophy and ridging of the tongue on the affected side during the postoperative period. This demonstrates that some of the central changes were reversible and that they were due to the hydrostatic pressure primarily, rather than to direct destruction, as one would ordinarily infer.

The observation that the arachnoid membrane was thickened and vascularized over the syrinx requires discussion. It will be noted that adhesive arachnoiditis was the third preoperative diagnosis. Inasmuch as the shocklike sensations occurred over a number of years, on moving the head, and the focal and general increase in pressure developed only within a few months of the time of operation, the arachnoiditis no doubt produced these shocklike sensations, which are typical of such a condition. Furthermore, the cerebellar tonsils were not then displaced into the canal, and their herniation cannot be used to account for these sensations referred downward into the extremities and upward from the neck on flexing the head. Although Nelson¹² and others have mentioned the presence of arachnoidal adhesions in certain cases of status dysraphicus associated with underlying cavitation, in the present case there is no probability that the arachnoidal changes played any part in the production of the cavitation. The cyst was primarily in the medulla, while the adhesions were atlanto-occipital. They were not adhesions of the massive and constricting type which sometimes surround the spinal cord and produce transverse pressure syndromes. This point is worthy of some emphasis because myelopathy and arachnoidal adhesions are not infrequent coincidental occurrences, but the adhesions should not be considered the etiologic factor in the myelopathy unless they are truly bandlike and constrictive.

GENERAL COMMENT

The conditions of syringomyelia, syringobulbia and syringopontia are merely topographically different expressions of the same basic

pathologic process. They are due to anomalous congenital dysplasia of ectopic gliogenous cell rests within the neuraxis, and the symptoms are caused by the sequelae of the developmental, maturative and degenerative processes which these undergo. Tamaki and Lubin¹³ have demonstrated islands and strands of primitive spongioblasts which proliferate and develop into adult glial tissue. This tissue, with its surrounding abnormal vasculature, undergoes a cycle of degeneration, necrosis and liquefaction as the patient reaches adult life, or often earlier. The cavitation may be single, multilocular or truly syringoid, extending for long distances within the neuraxis. The symptoms are produced in part by direct involvement of nuclear masses or fiber tracts, whose normal location has been supplanted by the hyperplastic cell rests. In part, they arise from disturbance of vascular supply to the normal nerve tissues due to the degeneration and diapedesis and hemorrhage which may occur in the abnormal vessels surrounding the hyperplastic zone. Also in great part, the symptoms are produced by direct pressure on adjacent nerve tissue by the collection of fluid under increased tension within the cavitations. As in the cases cited here and in those reported by other authors in which surgical intervention was beneficial, the relief of this tension resulted in arrest of the progress of the disease, and even recovery of some of the neurologic deficits. In neither of the present cases was any attempt made to obliterate the cavity or to affix a nonabsorbable foreign body to the edges of the myelotomy wound in order to maintain a communication between it and the subarachnoid space. Frazier,³ in a case with recurrent symptoms, operated a second time and found that the cavity had closed up and refilled with fluid. The second operation benefited the patient materially. In analysis of the cases which he reviewed, Frazier inferred that a technic to keep the cavity open yields the best results. Yet, in examining these results, one finds that in 8 of 16 cases an attempt was made to effect drainage by introducing a foreign body. In 3 cases there was "very little change." In 4 cases "moderate improvement" occurred. In 1 case there was "marked improvement." In 8 other cases drains were not inserted. Of these, "moderate improvement" occurred in 1 and "marked improvement" in 7. Thus, the figures cited by him and the inference drawn do not seem to be in accord, and more of the patients without drains were able to return to work than of the others. Davis¹⁴ cited 10 cases, in which immediate benefit resulted from the operation, although no patient was made free

13. Tamaki, K., and Lubin, A. J.: Pathogenesis of Syringomyelia: Case Illustrating the Process of Cavity Formation from Embryonic Cell Rests, *Arch. Neurol. & Psychiat.* **40**:748-761 (Oct.) 1938.

14. Davis, L.: *The Principles of Neurological Surgery*, ed. 2, Philadelphia, Lea & Febiger, 1942.

from symptoms. He was equivocal concerning the value of silver clips introduced into the edges of the myelotomy wound in an effort to maintain drainage. The value of introducing a foreign body is doubtful. Not only will nature attempt to close the defect but will also enclose the foreign body with tissue. Furthermore, the production of fluid within the syrinx is not a secretory function. It results from degenerative liquefaction of a mass of abnormal cells which have become inadequately nourished through abnormal blood supply. In my opinion, in most of the cases in which the symptoms demand operation an end stage in the pathologic process will have been reached, and recurrence at the point of operation will not take place.

SUMMARY

A case of syringopontia and one of syringobulbia, with direct surgical operation on the pons and the medulla, respectively, are reported, with successful outcome.

In both cases the condition simulated neoplasm clinically, in the relentless progression of symptoms.

In both cases the syndrome of advancing intracranial pressure was relieved.

In the case of syringobulbia the patient recovered from the neurologic deficits and was enabled to earn her own livelihood.

CONCLUSIONS

Syringoid myelodysplasias of the brain stem are amenable to surgical intervention.

Operation should be undertaken when the course is relentlessly progressive and the pace of development indicates advancing intracranial compression.

Neurological Institute of New York.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Physiology and Biochemistry

STUDIES OF DISTRIBUTION OF POLIOMYELITIS VIRUS. H. E. PEARSON and R. C. RENDTORFF, *Am. J. Hyg.* **41**:164, 179 and 188 (March) 1945.

Pearson and Rendtorff's first study of the distribution of poliomyelitis virus was concerned with the environment of sporadic cases. Testing stool specimens from almost the entire population of a village where poliomyelitis occurred in an adult, they recovered virus only from the 6 year old son of the patient. Pools of specimens from 127 persons in 39 families were uniformly negative. Of 30 persons associated with a patient with poliomyelitis in a small town, virus was recovered only from the stools of a sibling aged 5 years and from 2 playmates, aged 7 and 5 years, of another family. No virus was detected in the stools of the associates of 2 patients on farms or of 2 patients in a small town. Virus was not recovered from fecal specimens from farm animals or from flies, mosquitoes or the brains and intestines of rats or mice from the environment of these sporadic cases. In the second study stool samples from nearly all the children under 16 years of age were tested for the presence of virus after the occurrence of 2 cases of poliomyelitis in a small town. Members of the families of the patients also were tested. Of 282 persons in 146 families, virus was recovered from the brother and a group of 3 cousins of one of the patients and from children of 8 other families; 5 of these children were 2 years of age. From the degree of association of those found to harbor the virus, it was concluded that personal association was the principal factor involved in the spread of infection within the community. The third study concerned the distribution of virus in a selected district of Fort Worth during the 1943 epidemic of poliomyelitis. Stools from 524 persons were tested for virus by inoculation of monkeys. Six of 8 households representing 27 familial contacts were positive for virus, as were 8 of 45 households containing 80 nonfamilial contacts and 2 of 127 households representing 374 noncontacts. Virus was harbored by adults in 5 of the 6 positive households of familial contacts. Virus was not recovered from specimens of water, sewage, flies, ants, cockroaches or droppings of domestic animals. An agent that produced paralysis in mice and cotton rats was obtained from a pool of brains and intestines from 22 rats from the city dump. No virus was recovered from several batches of mice and rats collected in various other parts of the city.

J. A. M. A.

REFRIGERATION ANESTHESIA FOR AMPUTATIONS. SERGEI S. YUDIN, *Am. Rev. Soviet Med.* **2**:4 (Oct.) 1944.

Yudin discusses his observations on 106 patients. A total of 120 amputations of the extremities were performed under refrigeration anesthesia. The anesthesia was completely effective in 81 cases and satisfactory in 22 cases. No supplementary anesthesia was needed. In the remaining 3 cases the anesthesia failed. In 1 case the tourniquet became loose; in the second the patient withdrew the leg from the ice, and in the third an attempt was made to refrigerate the leg without a tourniquet. No anesthesia was obtained, and ether was administered.

Data are presented which indicate that refrigeration anesthesia does not diminish the viability of tissues at the amputation stump, nor does it delay healing. The reduced volume of blood circulating through the extremity and the lowered cell metabolism account for the excellent results.

Refrigeration anesthesia possesses definite advantages. It does not introduce an additional toxic agent into the body. The anesthesia is absolutely complete in that it eliminates the conduction of all painful, tactile and other stimuli along the spinal and sympathetic nerve paths. The progressive warming of the stump after the operation gradually restores conductivity in the severed nerves and also provides against excessive stimulation of the receptor centers in the brain; this occurs when the paralyzing influences of local or general anesthesia are removed suddenly. Finally, peripheral vascular collapse is prevented by the retarded absorption of toxins from the stump.

GUTTMAN, Philadelphia.

ADAPTATION TO ANOXIA AT DIFFERENT AGE LEVELS. I. A. ARSHAVSKI, *Am. Rev. Soviet Med.* 2:508 (Aug.) 1945.

Arshavski reports observations on the effect of anoxia on puppies of different ages and on adult dogs. Records were made of the reactions of the cardiovascular and respiratory systems and of the changes in hemoglobin and the erythrocyte count of the blood. In acute experiments, animals of different ages, beginning with those 1 day old, inhaled gas mixtures with varying oxygen concentrations from a bag supplied with a valve. According to the results obtained, the animals may be divided into three age groups: those from 1 to 18 days old, those from 18 to 45 days old and those above 45 days of age, including adults.

The author concludes that the young animal is more susceptible to anoxemia than the adult.

Data obtained on animals were confirmed in experiments on young children. The significant effect of anoxia is disorder of muscular coordination. In adults anoxemia was followed by paralysis of the feet, then of the hands and, finally, of the head musculature. In all children examined, the capacity to support the head in a vertical position was lost as soon as cyanosis appeared. There was recovery within thirty minutes to an hour.

Arshavski concludes that "children and young puppies are much less resistant to moderate oxygen deficiency than are adults. The lower tolerance of the younger organism is due to lack of the adaptive reactions which ensure the resistance of the adult organism to anoxemia."

GUTTMAN, Philadelphia.

PERMEABILITY OF THE HEMATO-ENCEPHALIC BARRIER IN MASSIVE ARSENOTHERAPY. E. I. KRICHEVSKAYA and D. I. LASS, *Am. Rev. Soviet Med.* 3:38 (Oct.) 1945.

Investigations were carried out on 43 patients in an effort to study transfer of arsenic into the cerebrospinal fluid. The subjects included 7 patients with tabes, 3 patients with progressive paralysis, 15 patients with syphilis of the brain, 2 patients with syphilitic meningitis, 1 patient with syphilitic pachymeningitis, 3 patients with syphilitic myelitis, 5 patients with latent syphilis, 5 patients with epidemic chronic encephalitis, 1 patient with epilepsy and 1 patient with Friedreich-Marie disease.

In this study the amounts of arsenic in the blood and the cerebrospinal fluid were determined by the method of Wintersteiner, as modified by Krichevskaya. Ninety-two patients received massive arsenotherapy by the drip method. Arsenic was detected within thirty minutes to five hours after injection of arsphenamine. In none of 49 patients, or 53.3 per cent of those studied, was arsenic found after twenty-four hours. In patients who had inflammatory disease of the meninges, arsenic always penetrated into the cerebrospinal fluid.

The authors state that they established the permeability of the cerebrospinal fluid barrier in more than half the patients with primary and early secondary syphilis without any apparent pathologic changes in the membranes. They state that they "detected arsenic in the cerebrospinal fluid many days and even weeks after the termination of treatment." They present data which indicate that arsenic penetrated into the cerebrospinal fluid in 37 of 49 patients who received massive

arsenotherapy and who had a temperature reaction. They assert, "it appears, therefore, that in four-fifths of the patients the temperature was a factor that facilitated the penetration of the barrier."

GUTTMAN, Philadelphia.

OXIDATION OF PHOSPHOLIPID CATALYZED BY IRON COMPOUNDS WITH ASCORBIC ACID. K. A. C. ELLIOTT and B. LIBET, *J. Biol. Chem.* **152**:617, 1944.

The respiration of tissue suspensions of brain or liver was stimulated for thirty to forty minutes by the addition of small amounts of iron or an iron-protein complex. This stimulation is greatly increased by the addition of ascorbic acid. Ascorbic acid alone causes the respiration of hypotonic tissue suspensions to be better maintained but has no effect on isotonic suspensions. With purified mixed phospholipid from the brain or liver at a neutral hydrogen ion concentration, ascorbic acid causes some oxygen uptake. Ascorbic acid plus iron-protein compound produces rapid initial oxidation, the rate falling off with time. The activity of the system varies in a complex manner with varying concentrations of the components. Of brain phospholipids, only the cephalin fraction undergoes oxidation with iron and ascorbic acid. Linoleic and linolenic acids are oxidized, but more slowly than is mixed phospholipid or cephalin. No other reducing agent tried was able to replace ascorbic acid in the system. The effects of different iron-protein preparations are not proportional to their iron content. Ferritin is less active than ferrin. Hemoglobin in small amounts is destroyed by phospholipid and ascorbic acid, giving rise to an active catalyst of the oxidation. Cytochrome c and hemin are inactive. Cyanide causes little inhibition of, and maintains for a longer time, the effects of iron and ascorbic acid. Epinephrine and other phenols are strongly inhibitory. Serum is strongly inhibitory; its effect may be largely accounted for by inhibitory effects of calcium and of protein. Some amino acids, especially histidine, have pronounced stimulatory and subsequent inhibitory effects on the system.

PAGE, Cleveland.

STUDIES OF SYNCOPE: III. DIFFERENTIATION BETWEEN VASODEPRESSOR AND HYSTERICAL FAINTING. JOHN ROMANO and GEORGE L. ENGEL, *Psychosom. Med.* **7**:3 (Jan.) 1945.

Romano and Engel, on the basis of physiologic and psychologic differences, distinguish between fainting related to vegetative neurotic mechanisms and fainting as a hysterical conversion. Fainting of the first type, they believe, is a vaso-depressor syncope, in which the emotional experiences are accompanied by changes in the circulatory system which lead to a loss of consciousness. This is shown by pallor, sweating, changes in the respiration and pulse and falling blood pressure, so that eventually cerebral anemia and marked distortion of electrical activity of the brain occur. In hysterical patients who experience fainting as a conversion symptom there is a notable lack of change in respiration, circulatory dynamics and electrical activity of the brain during the period of unconsciousness.

WERMUTH, Philadelphia.

THE CLINICAL CHARACTERS OF PAIN. W. GANADO, *Brit. M. J.* **1**:141 (Feb. 3) 1945.

Ganado describes pain as a group of distinct feelings, each of which has clinical characteristics of its own. The severity of pain depends on the tissues affected and the personality of the person involved. Also dependent on the tissues implicated are the power of discrimination, the power of location, the types of reflexes provoked and the quality of pain. According to Ganado, at least three types of somatic pain can be distinguished: (1) surface pain, from the cutaneous and mucosal surfaces; (2) subsurface, or intermediate, pain, from the subcutaneous tissues (where these are thin), from the submucosae and from some adjacent

structures, when the subcutaneous tissue is very thin, and (3) deep pain, from muscles and from all the sensitive deep tissues in general. These types are discussed somewhat at length.

ECHOLS, New Orleans.

Psychiatry and Psychopathology

A PSYCHIATRIC ADVENTURE IN COMPARATIVE PATHOPHYSIOLOGY OF THE INFANT AND ADULT. JOSEPH J. MICHAELS, *J. Nerv. & Ment. Dis.* **100:49** (July) 1944.

Michaels discusses certain broad psychobiologic principles, such as the concepts of repression and regression, of evolution and dissolution of the central nervous system and of the evolutionary process in general, and gives examples of their application in human behavior. In the same way in which certain infantile characteristics, such as enuresis and thumb sucking, may persist into adult life, so may some so-called functional symptoms of a psychosomatic disorder express themselves in a manner which utilizes modes of reacting which are consonant with and characteristic of earlier age periods. Adult visceral somatic dysfunctions may represent a recrudescence of infantile visceral somatic functions. This is well illustrated in anxiety states, in which the rapid respiration and pulse rates and frequency of urination and defecation are conditions which occur in the healthy, physiologic state of infancy. Child's concept of gradients is used to explain the isolated and autonomous action of a function which has become split off to appear as a visceral somatic symptom. Just as certain behavior patterns established in childhood may, under stress, become manifest in adult life, so may infantile physiologic patterns again become dominant over those acquired later, thus reversing the normal sequential series of gradients, in which an earlier life period paces a later one. In infancy anxiety is represented by a generalized startle response, which is the basic reaction to the physiologic and psychologic separation from the mother.

A high degree of homeostatic lability and a variable and inconstant adaptation to both internal and external stimuli are characteristic of infancy, just as a stable and delicately adjustable internal environment is an adult characteristic. Thus, heat regulation, water and mineral balance and the levels of red and white blood cells are all subject to oscillations in infancy, which are not found in normal adult life but which do occur in neurotic persons who are exhibiting autonomic and endocrine infantilism. Similarly, the symptoms and signs of the effort syndrome may be regarded in the sense of a reversion to the infantile condition, in which high and variable respiratory and cardiac rates are usual. The diarrhea, urinary frequency, blushing and cutaneous eruptions of the neurotic adult are other examples of this type of reversion.

The choice of the organ system affected by the psychosomatic disorder may be determined by its fusion with an emotional process at a labile period, when the former had not attained its full maturation.

CHODOFF, Langley Field, Va.

WAR AND ITS PSYCHIATRIC PROBLEMS. HOWARD P. ROME, *J. Nerv. & Ment. Dis.* **101:445** (May) 1945.

Rome points out that one effect of war has been to direct psychiatric thinking to the problems of the group rather than to those of the individual. Group and unit action has its principles and rules which cannot be explained by a mere quantitation of the individuals comprising the group. Precise answers are lacking to such questions as the reason for the formation and the nature of groups; what makes a group split, and along what lines, and how can group dissolution be prevented or brought about. Knowledge of these matters is of great importance both in psychologic warfare and in peacetime politics. Examples of the application of group technics in the recent war are the prophylactic value of battle training and inoculation and the success of group psychotherapy.

Returned service men will constitute a problem in group psychiatry. They will probably break up into three predictable segments—a "lost generation" portion, the chronically aggressive group and the comparatively normal and easily readjustable majority.

CHODOFF, Langley Field, Va.

SOME NEUROLOGICAL AND NEUROVEGETATIVE PHENOMENA OCCURRING DURING AND AFTER ELECTROSHOCK. H. H. FLEISCHHACKER, *J. Nerv. & Ment. Dis.* **102**: 185 (Aug.) 1945.

Fleischhacker reports observations on the physical phenomena occurring during and after electrically produced convulsions in over 70 patients, most of whom were women. The average voltage used was 95 to 120 milliamperes against a resistance of several hundred to 1,000 or more ohms for an average time of 0.2 to 0.4 second.

According to the strength of the stimulus, three levels of petit mal phenomena can be differentiated. These consist of petit mal proper; affective petit mal, characterized by emotional confusion with laughing or crying or moaning, and motor petit mal, characterized by movements, which may be (a) either "coordinate" or "pseudointentional" or (b) "organic," striatal, and then parakinetic, athetoid or jerking in type.

Some of the neurologic phenomena observed during the fit may be influenced by the position in which the electrodes are applied. If one electrode is placed nearer the external acoustic meatus than the other, asymmetric turning or convulsive movements may ensue.

Vegetative and vascular phenomena noted included the occurrence of cutis anserina, sweating and, rarely, dermographism. The pulse rate rises at the beginning of the fit, may increase or decrease toward the end and usually remains increased for a time after the fit. In some cases a pulse cannot be obtained from the radial or the carotid artery when it is still present in the abdominal aorta.

Through its influence on the diencephalon and, through this, on the pituitary and other endocrine glands, shock treatment has a definite neuroendocrine effect. The appetite is usually stimulated; some patients experience difficulty in sleeping, and there is an irregular effect on menstruation. Ejaculation may occur in some male patients during or immediately after the fit.

No discernible change was noted in samples of cerebrospinal fluid of about 20 patients examined during and after the course of treatment.

CHODOFF, Langley Field, Va.

THERAPEUTIC EFFICACY OF ELECTROCONVULSIVE THERAPY. GEORGE H. ALEXANDER, *J. Nerv. & Ment. Dis.* **102**:221 (Sept.) 1945.

In a previous communication, Alexander pointed out the desirability of utilizing a specific time interval between the termination of shock therapy and the evaluation of results, in order to avoid ascribing the influence of other healing factors to the specific treatment. He proposes that a period of thirty days following the final coma or convulsive treatment should be selected as the upper limit of time during which remissions should be attributed to its specific influence. Classifying treatment as successful only when the patient was able to leave the hospital within thirty days after his last treatment, Alexander reports the results of electroconvulsive therapy in 100 consecutively treated patients and contrasts the results so obtained with those obtained in a group of patients in whom the criterion of successful therapy was the ability of the patient to leave the hospital at some time subsequent to treatment. Using the thirty day criterion, the therapeutic efficacy was 51 per cent for all psychoses, 41 per cent for involutional psychoses, 67 per cent for manic-depressive psychoses, 49 per cent for involutional and manic-depressive psychoses combined and 56 per cent for schizophrenic psychoses. In the group in which the time factor was not considered, the percentages

of successful results were as follows: 87 per cent for all psychoses, 84 per cent for involuntional psychoses, 93 per cent for manic-depressive psychoses, 87 per cent for involuntional and manic-depressive psychoses combined and 83 per cent for schizophrenic psychoses.

These results lend support to the belief that it is only in cases of mental illness in which recovery with other forms of therapy is possible that electroconvulsive therapy hastens the process.

CHODOFF, Langley Field, Va.

OUTCOME IN DEMENTIA PRAECOX UNDER ELECTRIC SHOCK THERAPY AS RELATED TO MODE OF ONSET AND TO NUMBER OF CONVULSIONS INDUCED. LOUIS LOWINGER and JAMES N. HUDDLESON, *J. Nerv. & Ment. Dis.* **102**:243 (Sept.) 1945.

Lowinger and Huddleson report the relation of mode of onset and number of convulsions in 232 young male patients (95 per cent of them veterans of World War II) treated with electric shock. There were remissions in 54 per cent after a postshock period averaging three and a half months when the duration of the psychosis had been less than six months before the start of treatment. This 54 per cent breaks down into 55 and 50 per cent for cases of acute and insidious onset, respectively. The difference according to onset increases with the rise in the preshock duration of the psychosis. For a duration of twelve to twenty-three months the remission rates differed greatly, being 21 and 8 per cent for acute and insidious onsets, respectively. There were no remissions after a preshock duration of two years or over. Regardless of duration, the remission rate for the acute onset was 34 per cent; for the insidious onset it was 15 per cent.

For all preshock durations of over six months, courses of sixteen to twenty grand mal treatments yielded no better remission rates than courses of ten to fifteen treatments. For a duration of under six months, superior remission rates were obtained with ten to fifteen grand mal treatments. The incidence of improvement for all durations was slightly better with sixteen to twenty than with ten to fifteen grand mal treatments. Two courses totaling over twenty convulsions were practically useless.

CHODOFF, Langley Field, Va.

OBJECTIVE PERSONALITY STUDIES IN MIGRAINE BY MEANS OF THE RORSCHACH METHOD. W. DONALD ROSS and FRANCIS L. McNAUGHTON, *Psychosom. Med.* **7**:73 (March) 1945.

Ross and McNaughton made Rorschach records of 50 patients with migraine headaches and compared them with the records of 10 patients with headaches of undoubtedly psychogenic type, 15 patients with headaches of unknown cause, 55 symptom-free persons of superior intelligence, 50 miscellaneous psychoneurotic patients and 24 patients with tumor or injury of the brain. The records were analyzed only for objectively defined signs and were interpreted as a group rather than individually. The Rorschach method showed that the following personality features were associated with migraine: persistence toward success, difficulty in sexual adjustment, perfectionism, conventionality, intolerance and, in general, obsessive-compulsive features.

Patients with migraine show some of the characteristics of patients with psychoneuroses and cerebral disease but do not resemble either of these groups in their most characteristic composite Rorschach ratings.

WERMUTH, Philadelphia.

THE RORSCHACH PERFORMANCE WITH NEUROCIRCULATORY ASTHENIA. W. D. ROSS, *Psychosom. Med.* **7**:80 (March) 1945.

Ross studied 50 persons with neurocirculatory asthenia by the quantitative Rorschach method and compared the results with those for several control groups. The personality features of the patients with neurocirculatory asthenia included a tendency to give up easily under stress and an obsessional conscientiousness,

which made their problems appear unduly difficult to them. The patients with longer-standing conditions possessed neurotic features to a greater degree, while the patients whose disturbance was of recent onset showed more obsessional characteristics and in this respect resembled in personality the patients with migraine, as reported in a similar study.

WERMUTH, Philadelphia.

ELECTRONARCOSIS: CLINICAL COMPARISON WITH ELECTROSHOCK. GEORGE N. THOMPSON, JAMES E. MCGINNIS, A. VAN HARREVELD, C. A. G. WIERSMA and ESTHER BOGEN TIETZ, War Med. 6:158 (Sept.) 1944.

In the standardized electronarcosis technic an electronic instrument is used. This delivers a 60 cycle alternating current, which automatically compensates for moderate changes in resistance of the patient's circuit. A current of 160 to 250 milliamperes is applied through electrodes placed bitemporally (in terms of cerebral topography, bifrontally). The current is maintained at the initial level for thirty seconds, during which the patient has a tonic spasm; at the end of thirty seconds the current is decreased to 60 or 75 milliamperes, and at this time the patient usually shows a few mild clonic contractions. After sixty to seventy-five seconds the current is raised gradually at the rate of 5 milliamperes every fifteen seconds, to a maximum of 125 milliamperes at the end of five minutes. For reasons of standardization the treatment is usually terminated at the end of seven minutes.

The authors compared the cardiovascular responses, respiration, autonomic responses, ocular reflexes, tonic reflexes, pathologic reflexes and certain other abnormal neurologic signs in 50 patients seen during electronarcosis with those in 50 patients treated with electroshock and with those in 4 patients treated with a 200 milliampere current for thirty seconds.

In all three groups there was an initial cardiac arrest, lasting from three to seven seconds. Tachycardia began in electroshock after twenty seconds and returned to normal in a few minutes. In long-continued electroshock and in electronarcosis it began after thirty seconds and lasted throughout the shock, continuing at times for fifteen to twenty minutes.

In standard electroshock and in long-continued electroshock the blood pressure rose and returned to normal in three seconds. In electronarcosis the blood pressure returned to a level of 140 to 160 systolic and 90 to 110 diastolic during the first sixty seconds. It then began to climb, reaching levels of 170 to 220 systolic and 100 to 120 diastolic, where it remained until the current was cut.

In electronarcosis deep respirations continued for the duration of the treatment and maintained their depth and rhythmicity. In electroshock the respirations became more and more shallow during the first five to seven minutes after the current was stopped. In electronarcosis respiratory stridor occurred as the current was increased.

With all types of treatment autonomic flushing of the skin and pilomotor reactions occurred, but pilomotor contractions existed throughout the electronarcosis treatment. Sweating was more common with electronarcosis. In electroshock pupillary reflex to light was present, but in electronarcosis the pupils became miotic and remained fixed to light throughout the treatment.

In electronarcosis prolonged flexor tone and forced grasping were observed. These occur during deep insulin coma but are not present in electroshock.

The authors conclude that although electronarcosis has been used as an anesthetic agent for animals in its present status it is not practical as an anesthetic device for human beings.

PEARSON, Philadelphia.

WAR NEUROSES: PSYCHIATRIC EXPERIENCES AND MANAGEMENT ON A PACIFIC ISLAND. MEYER A. ZELIGS, War Med. 6:166 (Sept.) 1944.

In the new type of interisland warfare that was waged in the Pacific, fatigue, disturbed sleep, exposure to repeated noise or blast concussion and harassment

by the enemy are the common factors which contribute to the ultimate breakdown of previously stable persons when they are placed in a combat area. When to these are added the factors of geographic isolation, confinement to a small area, humid climate, lack of recreational facilities, no opportunity for leave and inability to retaliate or to unleash emotionally aggressive drives, there will be an increasing number of cases of neurosis.

Because of the locale and the particular factors involved, indications for evacuation of patients suffering from neurosis will differ from those which obtain in large scale land operations. Periods of duty in such theaters must necessarily be lessened, and rotation of personnel to different stations within the same area should be made possible if the incidence of operational fatigue and war neurosis is to be kept low. Effective psychotherapy of patients in the field is at best a difficult, and often a futile, procedure. The disheartening fact that less than 10 per cent of patients in this group improved when retained in the combat area forms the basis for this belief. From this limited experience it follows that prompt evacuation to a secure area constitutes one of the first essential steps in the treatment of such casualties. What the further disposition should be will depend to a great extent on the age, symptoms and response to early treatment. In general, persons who are older, married and with children will benefit more by return to their families, while the younger, more dependent, ones may be better rehabilitated away from home.

PEARSON, Philadelphia.

A PSYCHOMETRIC STUDY OF SENILITY. H. HALSTEAD, *J. Ment. Sc.* **89**:363 (July-Oct.) 1943.

Halstead describes a tentative battery of twenty-five short tests for the measurement of senescence. The functions tested included the "primary mental abilities"—verbal comprehension, word fluency, space, number factor, memorizing and induction or reasoning. The scale was the result of the application of nearly eighty tests to senile patients. The tests were classified into three groups according to the degree of difficulty experienced by the senile subjects. The most difficult tests were those in which the subjects were required to break away from old mental habits and to adapt to unfamiliar situations—tests of recent memory (logical); of judgment, planning and reasoning, and tests embodying difficult or lengthy instructions. Less difficulty was found with such tests as rote memory, fluency of associations, simple arithmetic and vocabulary. Performance was least affected on tests of visual recognition, old mental habits and simple motor tasks.

The tests for senility should be short because of straying attention, impaired comprehension and short retention. Perseveration is greatly increased in senescence. There was loss of steadiness and speed on the motor side.

The present mental status of the patients was estimated on the basis of eleven tests scored on the mental age method; the average mental age was between 10 and 11. An estimate of the former mental status of each patient was made by means of efficiency quotients on part of the Bellevue scale. A table comparing the present and the former mental status is shown.

KATZ, Boston.

PSYCHOSOMATIC CASUALTIES IN THE MIDDLE EAST. ALFRED TORRIE, *Lancet* **1**:139 (Jan. 29) 1944.

Torrie discusses 2,500 patients treated in a psychiatric base hospital in North Africa during the retreat from El Alamein and the advance from there to Tripolitania. He says he uses "the term psychosomatic deliberately in place of psychiatric and in a wider sense than is usual to emphasize body-mind unity." The diagnoses, in their order of frequency, were: anxiety neurosis, hysteria, psychopathic personality, endogenous depression, mental dulness, schizophrenia, organic states, paranoid states, reactive depression, neurasthenia, physical exhaustion, mania and obsessive-compulsive neurosis. The organic states were those the mental symptoms of which were secondary to or caused by a physical disability.

A higher proportion of casualties was found in "units with little cooperative life, such as small teams of drivers in the lines of communication." Married men showed a higher rate than single men. The author believes that irregular mail from home helps to make matters worse. The outstanding precipitating factor was an explosive battle episode. Change from a unit with good morale to one with lack of faith in the leader was an important factor also.

Of the first 1,201 patients discharged from the neurosis division, 51.6 per cent returned to full duty and 35.5 per cent to duty outside the battle area. Of these, 1,000 had anxiety neuroses and hysteria. Only 6 men had obsessional neuroses. The neuroses last mentioned, Torrie says, "are rarer than in civil practice because there is lessened guilt over unconscious aggressive impulses, these being sanctioned and indeed encouraged during wartime." Among these 1,000 patients headache was the prevalent symptom. It may have been physiologic at first, but later it became the main preoccupation. Anxiety, ranging from apprehension and fear to terror and panic, and tremors or tremulousness were next most common. It was often wise to have patients complaining of sleeplessness awakened now and again to show them that they slept well.

Of the 1,000 men with anxiety neuroses and hysteria, few had actual conversion hysteria, and 92 were subjected to rapid psychoanalysis. The main etiologic factor elicited was separation anxiety. In most cases a prototype for the adult reaction was found in the childhood history.

A ward environment was aimed at in which the soldier was surrounded by influences formerly lacking in his family situation, so that he could work out with the physician his conflict over aggression. "In treatment the purpose was to remove symptoms and bring the soldier back to his condition before illness and to remove, if possible, the underlying cause." Hypnosis, ordinary or chemical, helped to remove symptoms but was not so valuable in securing insight for the patient as for the physician. Prolonged narcosis, chiefly induced with diethylallylbarbiturate of diethylamine (somnifaine), was given to the patients with the severer illnesses, who were too acutely ill for other than physical approach. Of the 174 such patients, 30 to 40 per cent returned to full duty. Insulin and analeptic drugs and electric shock were used with good effect in some cases, and when indicated placebos were used in other cases.

The patients were discussed with the sister each morning, and her part as the "good mother" in charge of the ward was felt to be very important. Patients who did not show a recovery trend, and patients with conversion hysteria were felt to have a bad influence on other patients. The author believes a definite date of discharge should be given if possible, for it accelerates recovery in most patients, although it makes a few worse and thus indicates need for further treatment.

Psychotherapeutic interviews were given groups of 12 men at intervals. Hypothetic situations similar to their own personal difficulties were used, and afterward comments were encouraged from all. Group spirit was found to be poor at first. After the discussion each patient went to his daily job in the camp which he had chosen from a group of some twenty occupations. It was found that in occupational therapy the man in charge was more important than the materials used.

No illusions were held as to the chances of recurrence in the men. Patients treated with psychotherapy stayed on an average of eighteen and one-tenth days.

The author believes that civilian hospitals might benefit by making sure that neurotic patients up and about are actively employed.

MCCARTER, Philadelphia.

ACCIDENTS IN SHOCK THERAPY. CELSO PEREIRA DA SILVA and PAULO FERREIRA DE BARROS, Arq. assist. psicopat. estad. São Paulo 9:107 (March-June) 1944.

The authors report the occurrence of 16 accidents involving the bones and joints among 1,843 patients given shock therapy; 1,500 received metrazol shock

and 343 electric shock therapy. Three patients sustained fractures of the neck of both femurs; 2, unilateral fracture of the femur; 1, a fracture of the horizontal ramus of the os pubis, and 1, a fracture of the neck of the humerus; 1 had a comminuted fracture of the neck of the humerus with dislocation of the shoulder on the same side; 1 each, fracture of the clavicle, scapula and acromion process; 4, fractures of three or four dorsal vertebrae, and 1, a dislocation of the shoulder. Of these 16 patients with complicating accidents, 4 had hebephrenic schizophrenia, 1 the hebephrenocatatonic type and 1 the hebephrenoparanoid type; 1 had paranoid, 1 catatonic and 2 unclassified forms of schizophrenia; 2 had paraphrenia; 1 mental deficiency with a psychotic episode, and 1, hystericepilepsy. In 5 patients the injury occurred during the first treatment; in 5 others, during the second treatment; in 1, during the third treatment; in 2, during the fourth treatment, and in 2, during the fifth treatment. There were 13 complications among 1,500 patients treated with metrazol (0.86 per cent), and 3, among 343 patients treated with electric shock therapy (0.87 per cent). The youngest of the 16 patients was 21 years of age and the oldest 55; most of the patients were between 32 and 39 years old. Age did not seem to be a factor in the complications. The authors found no roentgenologic evidence of disease of the bone or of defective bone structure to indicate a predisposition to the complications. The duration of the illness of the patients treated also was not considered a significant factor. The authors believe that the type of restraint during treatment may have played a role. They do not, however, present any convincing statistics. Except for the bilateral fractures of the femurs, the prognoses for the skeletal complications were good.

SAVITSKY, New York.

Meninges and Blood Vessels

CEREBELLAR, DYSKINETIC AND ALTERNATING SENSORY SYNDROMES CAUSED BY ANEURYSM IN THE REGION OF THE PONS. VICENTE DIMITRI and JULIO ARANOVICH, *Rev. neurol. de Buenos Aires* 9:295 (Oct.-Dec.) 1944.

The authors report the third case of aneurysm of the superior cerebellar artery to be recorded. Bristowe reported a case in 1858 and MacSwiney a case in 1875.

A 30 year old farm laborer was admitted to the hospital five years after receiving a bullet wound; a bullet entered the region of the right ear and emerged from the left preauricular region. A few months later a sympathectomy was done for relief of exophthalmos on the right side. Diplopia and external strabismus of the right eye persisted. Four years after the injury the patient began to complain of dizziness and paresthesias on the left side of the body, with increasing difficulty in walking. Examination one year after admission showed bilateral exophthalmos, more prominent on the right side; paralysis of the right external rectus muscle, nystagmus, anisocoria, with sluggish reactions to light on both sides, and atrophy of both optic nerves with retinal hemorrhages. There was marked diminution of visual acuity, which rapidly progressed to blindness. Weakness of the lower left side of the face and nasal dysarthria were also noted.

The patient exhibited choreiform movements of both sides of the body while sitting. Tremors of the head and then of other parts of the body appeared especially with any kind of effort, such as talking or moving the head forward. The movements usually began in the head, extending to the rest of the body and ending in a series of violent contractions of the entire body. They were usually followed by a variable period of relative freedom from tremors. The slightest movement brought on similar dyskinetic phenomena. The patient was able to lie quietly on his back with the upper limbs semiflexed, his hands on his abdomen and the lower limbs in extension. On his attempting to change his position, the abnormal movements just described appeared. Pronounced dystonia was noted in all four limbs with passive movements, hypotonia alternating with fleeting increase in tonus. Passive movements of the joints and of the head also resulted

in coarse tremor. There were weakness and exaggeration of the tendon reflexes on the left side, and the abdominal and cremasteric reflexes were diminished, with a bilateral Babinski sign. There were bilateral marked cerebellar signs (dysmetria, heel to knee and finger to nose ataxia and adiadosokinesis), an alternating sensory syndrome on the right side of the face and left side of the body with a syringomyelic type of dissociation, left astereognosis and complete blindness due to secondary optic atrophy. The spinal fluid pressure was 70 (Claude), the albumin 40 mg., with 4 lymphocytes in the fluid. The patient died seven years after the onset of illness.

A large tumor was observed lying at the base of the brain, compressing the right side of the brain stem and extending rostrally from the level of the pontobulbar junction caudally to the bulbocerebellar angle. In its widest part it measured 5.3 cm.; the maximum anteroposterior dimension was 3.4 cm. The tumor was connected with the right superior cerebellar artery. There was displacement of the brain stem to the left, with extensive destruction of the right side of the base of the pons. Microscopic examination showed that the tumor was an aneurysm. Fragmentation of the media of the aneurysm with accumulation of iron pigment was considered evidence of trauma. Softenings were observed in the white matter of the right temporal lobe and in the right quadrilateral lobe. There was destruction of the greater part of the right middle peduncle, the homolateral restiform body, the right descending root of the fifth cranial nerve and the right spinocerebellar pathways and atrophy of the olives. The red nucleus was intact. The function of the medial fillet was apparently impaired by compression. There was also evidence of increased intracranial pressure, as seen in flattening of the convolutions and dilatation of the ventricles, probably due to closure of the aqueduct.

The unusual dyskinetic syndrome was considered a result of involvement of afferent and inhibition of efferent cerebellar pathways.

SAVITSKY, New York.

Diseases of the Brain

MIGRAINE HEADACHE: SOME CLINICAL OBSERVATIONS ON THE VASCULAR MECHANISM AND ITS CONTROL. MILES ATKINSON, *Ann. Int. Med.* **21**:990 (Dec.) 1944.

Atkinson reports his observations on 21 patients with uncomplicated migraine who were followed over periods varying from six months to two years. Each patient was subjected to an intradermal test with histamine. Negative results were reported for each patient. According to Atkinson, these observations indicate that "no case owned a primary vasodilator mechanism, that every one of the 21 cases of typical uncomplicated migraine owned a primary vasoconstrictor mechanism."

Atkinson administers nicotinic acid, for its vasodilator action, to migrainous patients. The regimen is as follows: From 25 to 30 mg. is injected intramuscularly in order to determine, by the extent of the flush reaction, the individual tolerance of the patient. From this reaction subsequent doses may be estimated, and a series of six to eight intravenous injections is given, starting with 20 to 30 mg. and increasing by daily increments of 5 mg. up to 50 mg. or such lower limits of tolerance as may be determined. A dose higher than 50 mg. is seldom required. After the course of intravenous injections the patient is taught to give himself intramuscular injections of such doses (25 to 50 mg.) and at such intervals (daily or three per week) as experience and the severity of the symptoms indicate. At the same time the drug is given in tablet form (50 to 150 mg. daily). After a period, which is determined by the clinical response to treatment, the patient is weaned from the injections and kept on a maintenance dose given by mouth. A high protein-low carbohydrate diet is recommended and advice given as to rest and

exercise and the beneficial effects on the vasomotor system of alternating warm and cool showers. Smoking is discountenanced and, when possible, stopped, on the grounds that migraine is a peripheral vascular disorder.

The results of this treatment were as follows: complete relief in 2 patients (followed over periods of four and six months); great improvement in 10 patients and moderate improvement in 5 patients. The treatment failed to relieve 4 patients.

Atkinson discusses the mechanism of the migraine syndrome. Migraine may be due to more than one cause. "It may be the result of such diverse conditions as vasospasm, allergy, exudative diathesis or endocrine disturbance."

The rationale for the use of nicotinic acid is the production of vasodilatation and thus an attack on the primary vasoconstriction which occurs prior to the onset of the headache.

GUTTMAN, Philadelphia.

ELECTROENCEPHALOGRAPHIC FINDINGS DURING AND AFTER ACUTE ENCEPHALITIS AND MENINGOENCEPHALITIS. IRA S. ROSS, *J. Nerv. & Ment. Dis.* **102**:172 (Aug.) 1945.

Ross investigated the electroencephalographic activity in 4 children during and after the acute stages of encephalitis. Gross disturbances were found in each patient. The abnormalities were generalized and consisted of slow, bilaterally synchronous waves of enlarged amplitude. The records were nonspecific, and the degree of the electroencephalographic disturbances was sometimes out of proportion to the mildness of the clinical symptoms. The disturbances in electrical activity of the brain may outlast the acute phase of the inflammatory process and in some cases may simulate the dysrhythmia associated with convulsive disorder, as well as the changes found in patients suffering from tumor of the brain.

CHODOFF, Langley Field, Va.

THE SYNDROME OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY RESULTING FROM A METASTATIC NEOPLASM. CHARLES DAVISON and LEO A. SPIEGEL, *J. Neuro-path. & Exper. Neurol.* **4**:172 (April) 1945.

Davison and Spiegel report their observations on 2 patients who presented neurologic evidence of the syndrome of the posterior inferior cerebellar artery. Both patients had primary carcinoma of the lung.

In the first case, the mental changes, which consisted of mood swings from naïveté and coyness to anger and aphasia, and signs referable to the pyramidal tract, which were more evident on the right side, were undoubtedly caused by a neoplasm in the left cerebral hemisphere. The Horner syndrome of the right, the sensory disturbances of the right side of the face and the contralateral sensory disturbances of the body, together with paralysis of the right side of the palate and uvula, were the result of a tumor which had metastasized to the lateral side of the medulla oblongata along the distribution of the posterior inferior cerebellar artery. In this case cerebellar signs could not be detected. Their absence remains unexplained, unless the signs of involvement of the pyramidal tract on that side interfered with the proper elicitation of indications of cerebellar involvement. There are occasional instances of occlusion of the posterior inferior cerebellar artery without cerebellar signs. In the present case the restiforme body was only slightly involved. The bilateral signs of damage to the pyramidal tract were readily explained by the lesion in the left cerebral hemisphere and that in the right cerebral peduncle. The sensory disturbances over the body below the ninth thoracic segment were best explained by the probability that not all the spinothalamic fibers were destroyed. The tumor cells in this case, and possibly in the second case as well, migrated from the lung by the blood stream and reached the posterior inferior cerebellar artery by way of the right vertebral artery, of which the former is a branch. The posterior inferior cerebellar artery may also take its origin from the basilar artery.

In the second case the clinical syndrome of the posterior inferior cerebellar artery was clearcut. The patient had cerebellar signs, which were lacking in the first case. It is believed that the dorsolateral part of the right side of the medulla oblongata was involved in this instance. No necropsy observations were available.

GUTTMAN, Philadelphia.

MILITARY ASPECTS OF NARCOLEPSY. MAX LEVIN, War Med. 6:162 (Sept.) 1944.

Twenty-five soldiers discovered asleep on post as sentinel were found to fall into three groups: 4 rebellious psychopathic men, who fell asleep intentionally; 19 good soldiers, who fell asleep because they more or less "carelessly" had failed to get enough sleep before going on post, and 2 men suffering from narcolepsy.

The military significance of narcolepsy is not limited to the fact that it is responsible for an occasional sentinel's falling asleep. There are other situations facing the soldier which appear to favor morbid sleep. These situations are comparable to the conditions which give rise to "inhibition" in the Pavlov experiments, and the tendency of men with narcolepsy (manifest or latent) to fall asleep in these situations supports the hypothesis that narcolepsy arises from undue "inhibability" (susceptibility to inhibition) of cerebral cells.

PEARSON, Philadelphia.

INTRACRANIAL ANEURYSM OF THE INTERNAL CAROTID ARTERY. JOSÉ M. MAINETTI and HUGO R. ORLANDI, Rev. med. d. Hosp. ital. de La Plata 1:19 (Oct.-Dec.) 1944.

Mainetti and Orlandi report the case of a woman aged 65 whose illness had begun a year prior to her admission with headaches, diplopia on right lateral gaze and dizziness. She continued to have recurrent severe pain around the right eye. Twenty-five days prior to admission to the hospital she was awakened by severe pain around the right eye, followed by headache and vomiting. Complete drooping of the right lid occurred at the same time. On admission, she also had dizziness, vomiting, a tendency to fall to either side while walking, hyperacusis and some difficulty with chewing. On examination, there were diminution of pain, touch and temperature sensations in the first division of the right fifth nerve, anesthesia of the right cornea, complete ptosis of the right eyelid and external and internal ophthalmoplegia on the right side. There were also definite involvement of the motor portion of the right cranial nerve, diminution of smell on the right side, diminution of pain in the anterior third of the right side of the tongue and labyrinthine hyperexcitability. The blood pressure was 190 systolic and 100 diastolic. The Wassermann reaction of the blood was negative. Examination of the spinal fluid showed a normal condition. An air encephalogram showed a mass pushing up the temporal horn of the lateral ventricle on the right and some displacement of the third ventricle. The preoperative diagnosis was neoplasm of the posterior fossa, probably in one of the cerebellopontile angles. No tumor was observed at operation. At autopsy an aneurysm of the intracranial portion of the right internal carotid artery was disclosed. Histologic studies of the aneurysm showed it to be of arteriosclerotic origin. There was no intracarotid injection of a contrast medium.

SAVITSKY, New York.

RELATION OF TRAUMA TO JUVENILE DEMENTIA PARALYTICA: CLINICOPATHOLOGIC AND MEDICOLEGAL STUDY. JOSÉ PEREYRA KÄFER, Rev. neurol. de Buenos Aires 9:117 (April-June) 1944.

Pereyra Käfer reports the case of a 12 year old boy, who was admitted to the hospital on May 18, 1937 and died Oct. 20, 1937. The father was alcoholic. There was no history of venereal disease. The patient began to talk at 1 year and to walk at 18 months of age. There was a definite history of retardation in school; his behavior was otherwise normal. The boy was able to shop in the neighborhood and to play, although he was nervous and at times difficult to manage. On June 6,

1935, when 10 years old, he was struck by a truck while crossing the street. There was a short period of unconsciousness; he sustained a small contusion in the left frontal region, an abrasion over the nose, a fracture in the middle third of the right tibia and a hematoma of the left eyelid. In four months he was entirely well and had no complaints.

In February 1937, about twenty months after the accident, he showed increased irritability, with progressively defective attention, pronounced alterations in conduct, disobedience, aggressiveness and impairment of speech. There was also gradual weakness of the left extremities. Two months later, in April 1937, he became incontinent. The pupils were unequal and fixed to light; there was weakness of the left side of the face, and spastic hemiplegia and hyperreflexia on the left side. The course was progressively downhill, and the patient died on October 20. The spinal fluid contained 18 lymphocytes per cubic millimeter and gave a 3 plus Pandy and a 3 plus Wassermann reaction. The Wassermann reaction of the blood was positive. Autopsy revealed the typical changes of dementia paralytica, with multiple scars in the convexity of the occipital lobes and in the base of the left frontal lobe, unquestionably of traumatic nature.

Although bridging symptoms were absent and the concussion was not very severe, the presence of the multiple traumatic scars makes it impossible to ignore the probable etiologic role of the head trauma, sustained two years before the onset of the dementia paralytica. Such traumatic scars can be present without clinical signs or symptoms. They create a locus minoris resistentiae in the brain.

SAVITSKY, New York.

STURGE-WEBER-DIMITRI DISEASE [NEVOID AMENTIA]. J. BEBIN, *Rev. de neuro-psiquiat.* 7:432, 1944.

Bebin states that Dimitri described the disease in 1923 independently of Weber, who recorded his case in 1922. Nussey and Miller collected 139 cases of the disease in 1939. The clinical picture of Sturge-Weber-Dimitri disease consists in facial nevi, glaucoma and signs of cerebral disease. Roentgenologic evidence of intracerebral calcification is usually present. Calcification may be within the brain substance or in the walls of the vessels, which are enlarged and thickened. The changes are most pronounced in the occipital lobes. The author reports 5 cases, without pathologic studies. In 1 case the nevus involved the distribution of the entire left trigeminal nerve; in another, only the distribution of the third division, and in 2 others, the area of the upper two divisions. In 1 case there was a small angioma near the angle of the mouth. All 5 patients had epilepsy and 2 gave a history of attacks of migraine. One had distal hemiatrophy of the left extremities, with hyperreflexia and a Babinski sign on that side. Two of the 5 patients had glaucoma. Only 1 patient showed intracranial calcification. None of the patients was treated surgically.

SAVITSKY, New York.

EPITHELIOMA OF THE CHOROID PLEXUS OF THE FOURTH VENTRICLE. JULIO ESPINOZA, RUBEN PERINO and H. VILCHES, *Rev. de psiquiat. y disc. conexas* 9:100, 1944.

The authors report a verified case of epithelioma of the choroid plexus of the fourth ventricle in a 12 year old boy. The child was admitted with a history of persistently severe headaches for seven months. Vomiting with accompanying exacerbations of headache, dizziness, bilateral tinnitus and diminution of vision had been present for a month. Examination showed diminished vision, bilateral papilledema, weakness of the right side of the face of mimetic type, cerebellar gait with dysmetria, impaired response to the Bárány test on the right side and counterclockwise nystagmus. Roentgenograms of the skull showed separation of sutures, erosion of the anterior clinoid processes and displacement and calcification of the pineal gland. Ventriculographic examination showed symmetrically dilated

lateral ventricles and dilatation of the aqueduct of Sylvius and of the fourth ventricle, which was pushed upward.

An epithelioma arising from the choroid plexus of the fourth ventricle was successfully removed. The patient did well except for transitory diminution of hearing and persistence of cerebellar signs two months after the operation. Vision improved.

SAVITSKY, New York.

CORTICAL DEAFNESS. J. LEMOYNE, *Ann. d'oto-laryng.* **10**:133 (Oct.-Dec.) 1944.

Lemoine says that the auditory tract is only partially crossed and that a unilateral temporal lesion does not impair hearing. The auditory tract has two relays and comprises three neurons. The first neuron runs from Corti's ganglion to the bulbar nuclei; this is the cochlear nerve. The second neuron, or the bulbo-diencephalic neuron, runs from the dorsal and ventral bulbar nuclei to the internal geniculate body. In this second segment the auditory tract is partly crossed. The crossed bundle comprises a superficial part (acoustic striae of the fourth ventricle) and a deep part, which at the level of the corpus trapezoideum constitutes the chief intercrossing of the auditory tract. The third neuron, the auditory radiations, runs from the internal geniculate body to the transverse temporal convolution, which comprises the center of the cortical projection of the auditory tract. This segment of the auditory tract assumes a special importance in the study of cortical deafness. The author presents the history of a patient with two associated types of disturbances: (1) severe bilateral hypacusia with dissociation of air and bone perception; (2) psychic deafness with total agnosia and disturbances of Wernicke's aphasia. He discusses the existence of cortical deafness and the differentiation of cortical deafness and bilateral labyrinthine deafness. The requirement of lesions involving the two transverse temporal convolutions explains the rarity of cortical deafness. A right temporal lesion has no effect; a left temporal lesion either has no effect or causes Wernicke's aphasia; in cases of bilateral temporal lesions either aphasia is evident or cortical deafness can pass unobserved and requires systematic investigations with the audiometer, or cortical deafness dominates but gives the impression of a labyrinthine deafness.

J. A. M. A.

Diseases of the Spinal Cord

POLIOMYELITIS AND RECENT TONSILLECTOMY. J. A. ANDERSON, *J. Pediat.* **27**:68 (July) 1945.

In 1943 Utah experienced the most severe epidemic of poliomyelitis in its history and had more cases per capita of population than any other state. Because of limited facilities for treatment of contagious diseases, only those patients were hospitalized who were reported by the local physician to have bulbar or respiratory involvement, to the exclusion of the patients with spinal involvement. This resulted in the admission of practically all the patients with clinically recognizable bulbar and respiratory forms. Of a total of 400 patients, 136 were hospitalized. The frequency with which a history of recent tonsillectomy was encountered in cases of the bulbar or bulbospinal type was the reason that a questionnaire was sent to 334 physicians in the state requesting the following information: (1) the number of tonsillectomies on children between 3 and 16 years of age done by them in July, August and September 1943; (2) the number of cases of poliomyelitis following recent tonsillectomy in these three months; (3) the age, sex and name of the child; (4) the interval between operation and the onset of symptoms; (5) the doctor's name, if he wished, or (6) the county in which he practiced. It was observed that 43 per cent of the cases of the bulbar and bulbospinal type were preceded by a tonsillectomy within thirty days of the onset. The incidence of poliomyelitis in recently tonsillectomized children was found to be 2.6 times as great as that in the general child population. The incidence of the bulbar and bulbospinal type of poliomyelitis was found to be sixteen times as great in recently tonsillectomized children as in the general child population.

J. A. M. A.

DERMATOLOGIC ASPECTS OF POLIOMYELITIS. J. G. REYES, New York State J. Med. **45**:1673 (Aug. 1) 1945.

During the recent poliomyelitis epidemic in New York city, Reyes observed 84 children, their ages varying from infancy to 14 years, admitted to St. Francis Hospital with the diagnosis of poliomyelitis. The disease was more common in boys than in girls, the ratio being 5 to 1. In 98 per cent of the children lesions were located on both infrapatellar areas, on the anterior and lateral aspects of both ankle joints, on the dorsa of both feet, on both soles and on the malleoli. The lesions were symmetric and were typically those of hyperkeratinization in the form of plaques, small papules or slightly verrucous elevations, with roughness and dryness of the skin of the legs. Lesions of these types and forms are encountered in cases of vitamin A deficiency. Their abundance seemed to be proportional to the severity of the poliomyelitic involvement, their prominence disappearing with the abatement of the disease. Among the children who were admitted for other lesions, only 1 in 8 had such cutaneous manifestations. From the extremely high incidence of these lesions of the skin in cases of poliomyelitis, Reyes deduces that vitamin A deficiency may be a predisposing factor of poliomyelitis. He advises that a diet rich in vitamin A should be given to all children, especially during the periods of epidemics, and this diet should be supplemented with cod liver oil or its concentrates. Since vitamin A deficiency will produce keratinization of the epithelium of the skin and the mucous membranes of the internal systems, it is possible that these structures which have been affected by the keratinizing metaplasia are open doors for the entrance of the poliomyelitis virus into the human body. It should be investigated whether the administration of vitamin A, either by mouth or parenterally, is of value in the treatment of poliomyelitis.

J. A. M. A.

SYNDROME OF AMYOTROPHIC LATERAL SCLEROSIS CAUSED BY CERVICAL "HOURLASS TUMOR." T. DE LEHOCZKY and L. PIRI, *Confinia neurol.* **6**:71, 1944.

De Lehoczky and Piri report the case of a neurogenous, epidural and paravertebral tumor in a boy aged 16 years. Two years prior to admission the boy had experienced pain in the neck and, later, pain in the left hand, with increasing weakness and paresthesia in the left arm and in the right leg. On admission, neither pain nor paresthesia was present, but the patient's strength was diminished and his muscles were atrophic. There was an egg-sized tumor in the left supraclavicular fossa, which could be traced to the left transverse process of the sixth and seventh cervical vertebrae. The sixth cervical intravertebral foramen was enlarged on the left side. The tumor, 9.5 cm. in length and 3.7 cm. in width, was removed in one piece through cervical approach without resort to laminectomy. On microscopic examination the tumor proved to be a typical neurilemmoma. It is suggested that the hourglass tumor gave rise to the symptoms by pressure of its intraspinal prolongation on the ventral part of the cord, the anterior pyramidal tract and the anterior gray horn cells. The removal of the tumor was followed by rapid improvement in the severe paralytic syndrome. This may be explained because of the epidural location of the intraspinal part of the tumor.

J. A. M. A.

HEINE-MEDIN DISEASE IN THE ADULT. CARLOS FONSO GANDOLFO, HUMBERTO R. RUGIERO, CARLOS CRIVELLARI and LEON CHAROSKY, *Prensa méd. argent.* **31**:1569 (Aug. 16) 1944.

Other investigators of poliomyelitis have emphasized the rarity of its occurrence in adults. The authors report 48 of 150 cases in patients over 14 years of age. Racial status, profession, sex and family history seemed to play no role in account-

ing for the appearance of the disease. The clinical picture differed in no way from that encountered in young patients; the spinal fluid findings were also the same. The authors did not note polynucleosis in the early stages of the disease, as reported by others.

SAVITSKY, New York.

SPONTANEOUS SUBARACHNOID HEMORRHAGE WITH PARAPLEGIA FOLLOWING INTRATHECAL TREATMENT WITH SULFAPYRIDINE. JUAN J. LAZARTE, *Rev. de neuro-psiquiat.* 7:355, 1944.

A half-breed chauffeur, aged 25, suddenly became dizzy and experienced severe pain in the back of the neck. He was able to drive his car home, though his vision became blurred. He lost consciousness soon after reaching home. On admission to the hospital, he had a stiff neck and other signs of meningeal irritation. Spinal puncture showed a bloody spinal fluid. At the time of puncture 2 Gm. of sodium sulfapyridine dissolved in 5 cc. of water was injected intrathecally. During the injection there were fleeting, repeated flexion movements of the trunk and tremulousness of the lower limbs. Neurologic examination on the second day showed extreme weakness of both lower limbs; there was absence of tactile sensation below the fourth dorsal segment, of pain sense below the third lumbar segment and of temperature sense below the tenth dorsal segment; deep sensibility was absent in the lower limbs; the knee and ankle jerks could not be obtained; there was urinary and rectal incontinence, and the cranial nerves and fundi were normal. The Kahn reactions of the blood and the spinal fluid were mildly positive.

The meningeal signs gradually disappeared. On July 12 examination showed notable improvement in motor power in the right leg; the sensory changes in this leg were gone; on the left there was diminution of sensation below the fourth lumbar dermatome; the sphincter disturbances persisted; there was still tendon areflexia and the spinal fluid was clear.

The author believes that the changes in the spinal cord were due to the intrathecal injection of sulfapyridine.

SAVITSKY, New York.

Peripheral and Cranial Nerves

INJURIES TO THE PERIPHERAL NERVOUS SYSTEM. HOWARD C. NAFFZIGER, *J. Nerv. & Ment. Dis.* 101:453 (May) 1945.

Failure of functional regeneration of divided nerves depends on many factors, including the nature of the nerve (regeneration of nerves of a purely motor or sensory type being better than mixed nerves), the condition of the muscles innervated, the nature of the injury (traction injuries offering a poorer prognosis), the presence or absence of infection and the site of division of the nerve. Injuries near the origin of long nerves require a long time for regeneration. Both experimental and clinical experience support the idea that suture after injury affords the best results provided the wound is clean and free from sepsis. These conditions are not usually met in cases of wartime wounds, so that delayed suture is often necessary. Suture can and should be performed, however, within two or three weeks of healing of the wound. In cases of nerve concussion and partial nerve lesions operative intervention may or may not be required, depending on the degree of improvement and the presence or absence of foreign bodies and persistent pain.

In order to provide the best possible care for victims of peripheral nerve injuries, Naffziger suggests that Army neurosurgical centers caring for patients with such injuries be placed near medical schools, so that experienced civilian neurosurgeons may collaborate in the treatment and opportunities for research may not be lost.

CHODOFF, Langley Field, Va.

EFFECT OF SULFONAMIDE DRUGS ON EXPERIMENTAL GUNSHOT WOUNDS OF PERIPHERAL NERVES. LOYAL DAVIS, GEORGE PERRET and WALTER CARROLL, War Med. 6:228 (Oct.) 1944.

The incidence of infection in wounds repaired twenty-four to forty-eight hours after injury was reduced from 83.3 per cent, in 6 animals, to 22.5 per cent, in 40 animals, when sulfathiazole jelly was introduced into the wound immediately after its receipt and a sulfonamide drug was powdered into the wound at the time of débridement.

No conclusive evidence was found that regeneration of nerve fibers, formation of axis-cylinders, myelination or absorption of decomposition products of myelin was changed with the use of the sulfonamide drugs. The deviation and abnormal distribution of regenerating nerve fibers were observed regardless of whether a strong mesodermal suture reaction or an epineural reaction was traumatic, infectious or caused by irritation from the sulfonamide drugs. The compounds did not interfere with the neurotization of autogenous or homogenous grafts or with the distal segment. However, in the series of animals receiving homogenous grafts a heteromorphous neurotization of the graft was found in all the animals treated with sulfonamide compounds, whereas similar grafts in untreated animals showed in all instances some degree of isomorphous neurotization.

The authors feel sure that if sulfathiazole jelly were introduced into extensive soft tissue wounds at the time of their receipt, so that all the ramifications of the wound were reached by the jelly, it would be possible to repair divided peripheral nerves at the time the soldier reaches a hospital where the wound could be carefully cleansed, trimmed surgically and sulfonamide drugs powdered into the wound. This would permit the repair of injuries of peripheral nerves at the earliest possible moment consistent with the best surgical treatment of fractures or other accompanying injuries.

PEARSON, Philadelphia.

THREE TYPES OF NERVE INJURY. H. J. SEDDON, Brain 66:237, 1943.

Seddon classifies peripheral nerve injuries as three types: (1) neurotmesis, in which all essential structures of the nerve have been severed; (2) axonotmesis, in which the axons are so severely damaged that complete peripheral degeneration occurs, while the supporting structures of the nerve remain intact, and (3) neurapraxia, in which paralysis occurs in the absence of peripheral degeneration. The morphologic changes associated with neurotmesis and axonotmesis are understood, while those accompanying neurapraxia are unknown. These types of damage to the peripheral nerve may result from a variety of injuries, and the same trauma may produce more than one type of injury. The author studied 650 cases of peripheral nerve injury. Of this group, neurotmesis occurred in 113. Retrograde degeneration occurred in the central portion of the nerve and extended a distance of 1 to 2 cm. Axonal regeneration began almost immediately at the central margin of the degeneration. At the point of injury a hematoma occurred, which was transformed into fibrous tissue. Seddon noted the usual changes of wallerian degeneration in the peripheral stump but emphasized the conspicuous proliferation of Schwann cells at the cut surface; this he designated as glioma. Frequently the neuroma, composed of scar tissue and regenerating nerve fibers of the central stump, can be felt though the skin. The glioma is rarely palpable. The clinical signs were those of complete interruption of both motor and sensory functions. Spontaneous regeneration was both rare and incomplete.

Seddon studied 81 cases of axonotmesis. In these cases the clinical picture was again that of complete interruption, but recovery was spontaneous. Although recovery was slow, it was, nevertheless, faster than that occurring in cases of neurotmesis after suture of the nerve. The recovery was perfect. In most

instances axonotmesis resulted from blunt injury. Occasionally the injury produced a local swelling of the nerve trunk. In comparing the state of recovery after axonotmesis and that after neurotmesis, Seddon found that after axonotmesis the degree both of motor and of sensory return approached perfection, while after neurotmesis delicate sensory tests, such as two point discrimination, revealed the presence of permanent sensory deficit and motor testing showed that independent and synergic movements were permanently impaired. Seddon concluded that neither infection nor the state of the peripheral portion of the nerve was responsible for this difference in recovery but that the disorganization of the architecture of the nerve attending neurotmesis was the determining factor.

Seddon found three important points in the differential diagnosis of axonotmesis and neurotmesis: (1) the nature of the injury, closed fracture being a more common cause of axonotmesis and open fracture of neurotmesis; (2) the time at which evidence of regeneration should appear spontaneously (if there is no sign of return of function at that time the nerve should be explored), and (3) the appearance of the nerve when exposed at operation.

In cases of neurapraxia Seddon found that motor paralysis was usually complete and greater than the sensory deficit; muscular wasting was slight or not evident; electrical excitability of the muscles was unchanged; recovery of motor function was both rapid and complete; loss of tactile sense was more extensive in area and of greater duration than loss of pain sensation; deep sensation was notably impaired; mild irritative phenomena were common, and the recovery of sensation was rapid.

Seddon studied 36 cases of neurapraxia. In 10 instances the injury was instantaneous; in 13 cases it was due to prolonged compression, and in 3, to intermittent trauma. The average duration of paralysis was about ten weeks. The clinical picture was found to be one of complete motor and incomplete sensory paralysis, the differences between motor and sensory disturbances and in the involvement of various sensory modalities being correlated with the type of fiber mediating these functions and their various susceptibilities to compression. Recovery was spontaneous and complete. At no time was there alteration of electrical reactions.

Seddon emphasizes the occurrence of combinations of the three types of lesions of the peripheral nerves.

FORSTER, Philadelphia.

Society Transactions

ILLINOIS PSYCHIATRIC SOCIETY

John J. Madden, M.D., Presiding

Regular Meeting, Dec. 6, 1945

Peter Bassoe, M.D. DR. PERCIVAL BAILEY, Chicago.

This paper was published in the February 1946 issue of the ARCHIVES, page 140.

A Warning Concerning the Use of Curare in Convulsive Shock Treatment of Patients with Psychiatric Disorders Who May Have Myasthenia Gravis. DR. LEE M. EATON, Rochester, Minn.

Curare has played an interesting role in the history of myasthenia gravis. Realization of the fact that curarization produces a clinical picture resembling myasthenia gravis led Walker (*Lancet*. **1**:1200 [June 2] 1934) to try its antidote, physostigmine, in the treatment of this disease. She found it effective and soon used neostigmine with gratifying results (*Proc. Roy. Soc. Med.* **28**:759 [April] 1935). Today neostigmine (prostigmine) is the most effective drug available for the relief of the symptoms of myasthenia gravis.

Bennett and Cash (*ARCH. NEUROL. & PSYCHIAT.* **49**:537 [April] 1943), from their experience with curare as a preventive of traumatic complications in convulsive shock therapy, were stimulated to test the sensitivity of patients with myasthenia gravis to curare. They found that the weakness of such patients was greatly intensified by small amounts. Administration of doses only one tenth to one fortieth of those required to produce mild generalized curarization in a normal person resulted in striking exacerbation of symptoms. They then utilized this observation in proposing a diagnostic test for myasthenia gravis (*Dis. Nerv. System* **4**:299 [Oct.] 1943; *ARCH. NEUROL. & PSYCHIAT.* **49**:537 [April] 1943). From considerable experience with this test, my colleagues and I of the section of neurology of the Mayo Clinic attest to its great value. We find it of particular aid in discriminating between other asthenic states and myasthenia gravis.

Furthermore, this test is helpful in confirming the diagnosis in those patients whose symptoms are so mild that the response to injections of neostigmine is often equivocal. In general, we find that the quinine test (*Proc. Staff Meet., Mayo Clin.* **18**:230 [July 14] 1943) and the curare test are of approximately equal value in excluding or establishing the diagnosis of myasthenia gravis in that borderline group of cases in which the neostigmine test is least reliable.

It may be well to emphasize at this point that the quinine and curare tests should not be used with patients who are seriously weakened, since further weakening may result in death in spite of artificial respiration and administration of massive doses of neostigmine. A tragic personal experience with such a case, in which all the safeguards failed, allows me to speak with authority on this matter.

Experience with curare in cases of myasthenia gravis led me to predict that the ordinary curarization of a depressed patient, preliminary to convulsive shock therapy, might prove fatal should the patient also suffer from myasthenia gravis. Confirmation of the prediction was not made by experience because, fortunately, in the case to be reported the presence of mild myasthenia gravis associated with a depression was recognized before convulsive shock therapy for the depression was begun. A brief account of this case will prove instructive by emphasizing the dangers involved.

REPORT OF A CASE

A white man aged 36 came to the Mayo Clinic in March 1944 because of exhaustion, insomnia, inability to relax and a fear that he was soon to die. He stated that he worried about everything and admitted having thoughts of suicide. It was learned that for years he had experienced periods of depression, the most intense beginning in February 1938, as a reaction to a change in his work. Within ten months his condition had progressed to a point where he could work no longer as an office manager. As a result, he was confined to psychiatric hospitals for five months. The depressive symptoms became sufficiently mild by the summer of 1939 that he was able to return to work, and within a few months he felt well again.

The patient continued to feel well until the summer of 1943, when he noticed that he could not see clearly unless he held his head far back. Three weeks after he noticed this, diplopia began to occur occasionally and ptosis of the right eyelid became noticeable. At about the same time he began to have difficulty in sleeping, to worry and to feel much as he had during the previous depression. He consulted two neuropsychiatrists, one of whom gave him an injection of neostigmine. The diagnosis of myasthenia gravis was evidently dropped from consideration when the patient reported that after the injection he felt no better.

Examination at the clinic revealed slight psychomotor retardation and thought content appropriate to the depressed emotional state. Neurologic study revealed a moderate degree of ptosis of the right eyelid and considerable weakness of the superior rectus muscle of the right eye and of both anterior tibial muscles. The peroneal muscles and those extending the toes were perceptibly weaker than normal.

The diagnosis of myasthenia gravis on clinical grounds was substantiated when definite strengthening of the weakened muscles occurred after the patient received a subcutaneous injection of 1 mg. of neostigmine methylsulfate and when weakening of the muscles occurred after quinine sulfate was administered orally in two doses of 10 grains (0.65 Gm.) each two hours apart. Furthermore, 0.2 cc. of intocostrin, which contains 4 mg. of curare, produced a definite weakening effect. This dose is slightly less than one twentieth of the dose that would ordinarily be used preliminary to convulsive shock therapy for a man of his weight (184 pounds [83.5 Kg.]).

The diagnosis of manic-depressive psychosis and myasthenia gravis was made. It was suggested that the patient take 15 mg. of neostigmine bromide three to five times daily.

The patient returned two months later because of the persistence of psychiatric complaints. Myasthenia gravis was of mild degree, and the patient was well relieved when he was taking neostigmine. From May 26 to June 7, 1944 seven electroshock treatments were administered without preliminary curarization. Four resulted in convulsions. The patient was much improved and returned to work. He relapsed soon, and when he came back for further treatment, four more electrically induced convulsions were given. When he returned home, he refrained from work, at our insistence, for a period of two months. When last heard from, in July 1945, he was well except for ptosis of the right eyelid, which continued to be alleviated when he took neostigmine bromide, and he was successfully engaged in a new occupation.

COMMENT

Although this patient had myasthenia gravis in its mildest form, he was extremely sensitive to curare, as is usual in such cases. He suffered from a depressive reaction severe enough to warrant electroshock therapy, to which he responded well. Had the associated myasthenia gravis not been recognized and had an ordinary dose of curare been administered as a preliminary to electroconvulsive therapy, death probably would have resulted in spite of heroic measures to combat the effect of curare.

The value of convulsive therapy has become widely established in the treatment of psychiatric disorders, particularly depression. Furthermore, curarization preliminary to convulsive therapy is widely used. Stewart (*Dis. Nerv. System* 4:236 [Aug.] 1943) pointed out that in 1943 more than one hundred institutions in the United States had adopted this plan of treatment. Myasthenia gravis is not nearly so rare as it was once assumed to be. The recognition of milder atypical forms of the disease seems to account for the apparent increased frequency. For many years the diagnosis has been made in approximately 4 out of every 10,000 new cases encountered at the Mayo Clinic. In the last four years I, personally, have examined 175 patients who had myasthenia gravis. In this large group there was no more than an average proportion of major psychiatric disturbances. One patient had a manic-depressive psychosis; 1, a moderately severe reactive depression; 2, reactions resembling those of schizophrenia, with associated exophthalmic goiter; 1, a severe post-traumatic psychoneurosis with paranoid trend, and 1, frank conversion hysteria. Actually, only 2 patients of this group were given convulsive therapy, and for 1 other such treatment was considered. I realize that the specific set of circumstances, that is, a case of unrecognized myasthenia gravis plus associated psychiatric disorder for which convulsive treatment is given after preliminary curarization, rarely will be encountered. However, myasthenia gravis must be ruled out in any case in which curarization is to be used preliminary to electroshock if tragedy is to be avoided. It is hoped that this report may stimulate physicians who use the treatment to consider that myasthenia gravis might be present before proceeding with curarization. To imply that preliminary curarization is to be avoided in the ordinary case in which convulsive therapy is used is not my intention.

Psychiatric Overlay in Physical Disease. DR. LEO KAPLAN, Chicago.

This paper was given primarily to point out the many cases of functional overlay that were seen in the Army. The effects of psychogenic factors on physical disease were elaborated on, indicating the relation between psyche and soma. Some of the psychopathologic mechanisms which were apparently responsible for the prolonged delay in recovery from organic disease were pointed out.

A number of cases of organic disease with superimposed functional overlay were presented, including those with such diagnoses as traumatic vasospasm, acrocyanosis, thromboangiitis obliterans and motor weakness of various extremities associated with fracture and prolonged application of casts.

The types of treatment and the results were indicated, with formulation of the following conclusions: (1) motivation for production of functional overlay is the same in civilian life as in the Army, namely, gain to the patient by obtaining dependency, attention and security; (2) early recognition and differentiation of that portion of the illness which is functional and that which is organic in order to obtain a better prognosis for recovery; (3) early recognition of functional overlay to prevent chronic invalidism in veterans to be discharged from service.

Group Psychotherapy with Private Patients. DR. ABRAHAM A. LOW, Chicago.

A class was started in 1941 with a small group of private patients who met once a week, one patient being interviewed while the others listened. At present the class is attended by upward of 70 patients. The class consists in about equal parts of psychoneurotic and former psychiatric patients.

The discharged psychotic patient suffers from residual symptoms, such as tenseness, difficulty in sleeping, fatigue, numbness, pains, palpitation, blurred vision, head pressure and other sensations, fears and compulsions. These residual symptoms must be relieved without delay. Otherwise, apprehension on the part

of the ex-patient and his relatives creates an unhealthy atmosphere, which may precipitate a relapse.

The psychoneurotic patients admitted to the class are chiefly of the chronic type. After making the rounds of physicians and clinics, they have decided they cannot be cured. All they want is a prescription or a "pep talk" for temporary relief. Having discounted the possibility of a final cure, they cultivate a self-appointed defeatism.

The physician is ill equipped to deal with defeatism. To his endeavor to "sell" the idea of mental health the patient offers a sort of "sales resistance." The patient's credo is that the physician would not dare tell the truth even if he knew the condition was incurable. Medical ethics forbids frankness. In class sessions the chronic patient is made to listen to a "colleague" who has successfully "licked" his own chronicity. That colleague is convincing. He has nothing to "sell." The patients attend one weekly class conducted by the physician. In addition, they are encouraged to join the Recovery Association, in which the patients gather in weekly home meetings. There they hold panel discussions, encouraging one another to give up their resistance. This resistance is called "sabotage," because it sabotages the physician's authority. The most pernicious form of sabotage is self diagnosis and self prognosis. If the patient calls his compulsion "unbearable" or his fear "uncontrollable," he pronounces his difficulty serious (diagnosis) and difficult to repair (prognosis). With this, he sabotages the physician's authority.

To check defeatism, the patient must be taught how to curb his temper. Temper precipitates and intensifies symptoms. On the other hand, symptoms give rise to outbursts of temper. A vicious cycle is thus established. This must be broken by class instruction and the self-help activities of the Recovery Association.

The main advantage of the combined class and self-help method is to gain time for the physician. With the aid of various group psychotherapeutic technics it is possible for the physician to reduce the time spent on the individual patient during the office interview. In this manner, group psychotherapy is supplemental to individual psychotherapy.

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Joseph H. Globus, M.D., *President, New York Neurological Society, Presiding
Joint Meeting, Dec. 11, 1945*

Presidential Address: Brain Tumor: Its Future in the Light of the Past. DR. JOSEPH H. GLOBUS.

This paper was published in the April 1946 issue of the *Journal of Neuro-pathology and Experimental Neurology*, page 85, under the title "Brain Tumor: Its Contribution to Neurology in the Remote and Recent Past."

Suprasellar Calcified Lesion of Unusual Size: Report of a Case. DR. ALFRED GALLINEK.

An American-born white man aged 35, married, was first examined in October 1944 and has been under observation since that time. At the age of 32 the patient first experienced headaches, which since then have occurred intermittently. The average frequency has been twice a week. The headaches usually begin late in the afternoon. The patient soon discovered that he could eliminate the headache by lying down. He also noticed that he could aggravate the pain by stooping. The headache is always bifrontal and is localized in the orbits behind the eyes and across the bridge of the nose. Recently there have been several episodes of nausea and vomiting, which also were relieved by lying down. For the past two years there have been occasional photophobia and blurring of vision. At the age of 33

the patient experienced the first of a series of "weak spells." These spells occurred infrequently in the beginning, but during the past year he has had seven attacks of loss of tone. These attacks were initiated by a sensation of numbness in the forehead and on both sides of the face. This sensation was followed by flushing and sweating in these areas. Intense weakness then appeared in the legs, and about a minute after the onset of the initial numbness the patient fell to the ground, with perfect preservation of consciousness. During the past year the duration of the attacks of loss of tone has been about five minutes from the onset of the first symptoms until the moment when the patient is able to resume standing or walking.

Invariably, his headaches have been completely relieved by rest in a recumbent position for about fifteen to thirty minutes. His condition has not prevented him from working nine hours a day. The past history is noncontributory except for a head injury at the age of 6 years, when he fell out of a cherry tree, hitting his head on a rock. He does not recall any details but remembers that he did not have to be hospitalized. It is noteworthy that there is no history of polydipsia or polyuria, that the patient's sex life is normal and that he has to shave daily.

On examination he weighed 125 pounds (56.7 Kg.) and was 5 feet 6 inches (167.6 cm.) tall. He was of slight body build; no abnormalities were evident on general examination. He was right handed. Both disks showed slight edema (right eye, $\frac{1}{2}$ D.; left eye, 1 D.). There were blurring of the nasal margins and moderate temporal pallor. Both visual fields showed marked constriction. Visual acuity was 20/30 bilaterally. There were hippus and slight nystagmus on left lateral gaze. The cranial nerves were otherwise normal except for a slight rigidity of facial expression and vague asymmetry in the innervation of the left side of the mouth, which might represent weakness of the left side of the face of central type. Slightly rigid posture was noticeable, particularly on walking. An inconsistent tendency toward dorsiflexion of the big toe was occasionally observed on the left, as well as some fanning. There was downward drift of the left upper extremity, with convergence and pronation in the right upper extremity. The rest of the neurologic status was normal.

The initial spinal fluid pressure was 200 mm.; after withdrawal of 10 cc. the pressure was 170 mm. On subsequent spinal punctures, the initial pressure was 130 or 140 mm. and the final pressure 70 mm. Examination of the spinal fluid showed a total white cell count of 3 per cubic millimeter, a total protein of 160 mg. per hundred cubic centimeters, a normal colloidal gold curve and a negative Wassermann reaction. The Kline reaction of the blood was negative. The urine was normal. The blood sugar and the glucose tolerance curve were normal.

Posteroanterior and left lateral stereoscopic roentgenograms of the skull showed a large, circumscribed shadow of calcium of almost bony density lying in the suprasellar region, in the midline. The inferior margin was relatively straight. A small shadow of calcium was seen posterior to this large mass, apparently the posteriorly displaced pineal gland. The sella turcica was slightly enlarged. The posterior clinoid processes were atrophic. The bones of the vault showed no definite abnormalities.

The electroencephalogram was normal. The basal metabolic rate was -14 per cent. The results of other routine laboratory studies were normal.

In analysis of this case, the following groups of symptoms are distinguishable: intermittent headaches, to a great extent depending on and relieved by changes in posture; attacks of loss of tone in the lower extremities; vague parkinsonian features in facial expression and gait; a slightly low basal metabolic rate, and moderate papilledema, with constriction of the visual fields and moderate difficulties in vision.

The tumor in this case must occupy most of the region of the third ventricle and its adjacent structures. Tumors inside the third ventricle are known to produce few symptoms, but for tumors involving the third ventricle as well as adjacent structures Fulton and Bailey have enumerated not less than nine syndromes, most of which were not distinguishable in our case. Wilfred Harris stressed the importance of paroxysmal postural headaches as a diagnostic sign of intraventricular

cysts and tumors. Stookey described the same type of headache as that seen in the case presented and ascribed it to intermittent obstruction of the foramen of Monro by a neuroepithelial cyst. The mechanism of these headaches was explained by Harris as a block of the foramen of Monro. Considering the size of the lesion in the case presented, it is likely that compression of and relief of pressure on both veins of Galen play an important role.

Attacks of loss of tone were also seen by Harris in a case of colloidal cyst in the foramen of Monro. The affective component seen in most cases of idiopathic cataplexy was absent in the case presented.

It is difficult to decide whether the lesion in this case is a calcified meningioma (originating from the anterior portion of the falx), a craniopharyngioma or a dermoid. It was felt that not much additional information could be expected from air studies and that they might be dangerous. The problem of surgical approach was discussed seriously. In view of the size of the lesion and its localization, there is no doubt that surgical removal would be difficult. The fact that the patient is able to work and to lead a normal life favors a conservative attitude, particularly since all his subjective symptoms usually disappear for about two months after withdrawal of 10 to 15 cc. of spinal fluid.

The case is presented because of the striking discrepancy between the magnitude of the lesion and the comparatively mild character of the symptoms, as well as the diagnostic significance of postural headaches and attacks of loss of tone.

This paper was published in the May 15, 1946 issue of the *New York State Journal of Medicine*, page 1127, under the title, "Postural Headache and Attacks of Loss of Tone in a Case of Calcified Suprasellar Tumor."

DISCUSSION

DR. H. A. RILEY: This case of Dr. Gallinek's has been a subject of considerable interest to us at the Institute. Dr. Gallinek presented the case at one of our conferences, and we were all in doubt as to the actual nature and possible location of this bony growth. There is little for me to discuss in this presentation, for Dr. Gallinek has covered as adequately as is possible while the patient is still alive the clinical and supposed pathologic features of the situation. The two points of interest are those which he emphasized, the attacks and the paroxysmal headaches. The attacks are similar to cataleptic seizures but are entirely dissociated from any change in emotional tone. That leads me to believe that these attacks may be of cerebellar origin—attacks of cerebellar atony—for some of the neoplasms and lesions of the third ventricle produce predominantly cerebellar symptoms. The other feature, which is the presenting symptom in this patient, is the paroxysmal episode of headache. Dr. Gallinek has emphasized the two explanations for this condition: (1) an episodic block of the foramen of Monro and (2) the theory which seems less attractive to me, namely, interference with the veins of Galen. At their origin near the foramen of Monro the veins of Galen are very small, and they become large only as they receive successive additions—the vein of the corpus callosum, the vein of the septum pellucidum and the striatal and choroidal veins. They assume large proportions only as they pass back to form the great vein of Galen, which with the inferior longitudinal sinus and the basilar veins forms the straight sinus. It seems to me that the cause of this intermittent, episodic headache, which is positional in character, is much more likely to be blockage of the foramen of Monro, forming a hydrocephalus as a result of the damming back of the cerebrospinal fluid in the ventricular system.

I remember a dramatic case of what I thought at first was simple migraine. The patient came to me with the complaint of headache, which came on only in episodes, accompanied with some visual phenomena—streaks, jags and lines of light, which are frequently associated with ophthalmic migraine. His mother and several other members of the family had migraine. I kept him under observation for a year or so, treating him, as all try to treat migraine, without any great success. About two years after I first saw him, he returned with his headaches materially

increased. He had noticed that they occurred only after he had been studying (he was a student at Princeton), leaning forward over his desk, for a considerable length of time. Only at that time did he show any papilledema, with hemorrhages but no localizing signs. I sent him to the Neurological Institute, where an encephalogram demonstrated beautifully a colloid cyst of the third ventricle. Dr. Davidoff removed the growth by a transfrontal approach, with complete relief of the symptoms and cure of the condition, except that temporarily the patient had a series of convulsions, which were controlled with phenobarbital. I feel that the explanation of this type of headache is to be found in an obstruction of the foramen of Monro.

I believe with Dr. Gallinek that the location of this tumor and its probable origin are in the third ventricle. I think that one may conclude from the roentgenograms that it is too high in the cranium to be a suprasellar craniopharyngioma and too low to have its origin in the anterior portion of the falx; I believe it is too far caudal to arise from the falx, as this membrane arises from the crista galli. It is central in location and occupies the position of the third ventricle, which, if the tumor is so situated, must be greatly dilated.

The other questions of importance in this case concern the advisability of carrying out an air study and of operating. This man is doing fairly well; he has lived for a number of years with this neoplasm, and he probably will live comfortably and productively for a considerable time. I think it is better not to attempt any air studies or operation; a live patient is better than a dead certainty.

Every neurologist who has become accustomed to see cerebral neoplasms recognizes that practically no significance is to be attached to the size of a tumor so far as symptoms are concerned. Those who are entering the field of neurology must appreciate the fact that a tumor makes its presence known primarily, and almost exclusively, by the rapidity of its growth. If a tumor grows slowly, a brain can accommodate itself to the presence of a growth of almost any size; if it grows rapidly, a small tumor may produce serious, and evident and prompt, symptoms.

I wish to congratulate Dr. Gallinek on his careful study and the interesting presentation of this case.

DR. BYRON STOOKEY: I think Dr. Gallinek and Dr. Riley have said everything that is of interest. I am delighted that this patient is Dr. Gallinek's patient and not mine. I should like to thank Dr. Riley for looking at me when he said he would rather have a live patient than a dead certainty. Certainly, I think from the surgical standpoint the patient is better off out of the surgeon's hands. This man is well adjusted to his tumor; since it is calcified, it is no longer growing; he can be followed carefully clinically, and if an operation should be desirable it can be undertaken. I have an idea that the tumor could be removed by heroic measures, perhaps requiring amputation of the frontal lobe. I agree with Dr. Riley and Dr. Gallinek that the growth is probably in the third ventricle, so that an approach can be made if necessary.

Would Dr. Gallinek state what position the patient assumes when he is relieved of his headache? Most patients are relieved, not by lying with the occiput down, but by placing the forehead down. Many a sudden death has been attributed to cardiac disease which was actually due to a tumor of the third ventricle suddenly obstructing the foramen of Monro and producing a rapidly developing hydrocephalus. I think the explanation of obstruction of the ventricular system is the correct one.

DR. ALFRED GALLINEK: In answer to Dr. Stookey's question, the patient has to lie flat on his back to obtain relief from the pain.

Phyletic Manifestations and Reversions. DR. A. A. BRILL.

The author pointed out that both Freud and some of his pupils came to the realization that certain neurotic manifestations cannot be adequately explained by the patient's past but that they can be understood only on the basis of phylogenesis.

The author cited a case of a husband and wife who had lived together for almost a generation, constantly quarreling, separating and fighting. Their behavior was predominantly anal-sadistic; object libido seemed to have played only a minor part in their married existence. The author was struck by the fact that although both husband and wife belonged to a better than middle class cultural level, they continually hurled invectives at each other which referred to the posterior region. As these expressions have always been popular among persons of the lower social strata, it was concluded that they must conceal some long-forgotten pleasant outlets. In other words, the invitation to kiss one's behind, which is now disgusting, must have once formed a pleasant outlet.

The author then examined the biologic development of the penis and vagina and showed that they both stemmed from the anus, that they both developed from the cloaca, and that the rudimentary penis for the transmission of semen only is first encountered in the ornithorhynchus, which forms the link between the three great animal families. This explains why the anal functions still retain some of their erstwhile pleasurable feelings, why many people like to linger and read in water closets or in "comfort stations." They are unconsciously reliving some long-forgotten pleasures. The invitation hurled during fights and quarrels by people of the lower social levels has the same meaning. That these feelings have always existed in concealed form is readily demonstrated by customs and proverbs of primitive and modern peoples and by their occasional outcropping in the literature of the day. The author concludes that the tabooed invitation to kiss one's posterior is a revival of a phyletic engram in distorted form.

DISCUSSION

DR. EDWARD KEMPF, Wading River, N. Y.: Last summer Dr. Brill told me about his patient and his impressions; and I, in turn, told him I had been for several years engaged in an investigation of the evidence on the biology of bisexual differentiation and had found a good deal of information concerning man's recapitulations of the phylogeny of bisexual differentiation in his ancestors. When his paper was announced, I sent him a copy of my paper; so he promptly called me in to discuss his paper at this meeting.

I should like to make a few comments along this line, guided largely by Dr. Brill's interesting material and speculations. I think that the average physician in his education has not been taught to realize the profound importance of ontogenetic recapitulation in the development of man. There is an inclination to think of it as a recapitulation in the embryonic or prenatal stage only; actually, it goes through a long series of steps, which begin with the embryonic stages, including the cloacal differentiations of the anus and urethra, penis and vagina and the hermaphroditic differentiations. All these emerge before birth. After birth, apparently, the cerebral cortex is conditioned by the morphologic and physiologic processes of the organism, as well as by the environment. At birth the cerebral cortex is practically an unconditioned instrument. At this period the infant goes through the differentiation of the oral food-taking and finger-sucking reflexes and of the cystic-urethral-urinary and anal-rectal-defecating functions, combined with a great deal of exhibitionism and learning. It is just beginning to be realized that he should go through these phases, because they are extremely important as foundations for elaborating his future sociability. One finds in dogs, apes and monkeys such behavior in the young animal, which gradually disappears in the adult. The same process seems to occur in man, and all these recapitulations are found in primitive, as well as in civilized, man.

Primitive man is considerably more tolerant toward these functions than is civilized man. Primitive man, like civilized man, usually grades his compliments to another in terms of what is beautiful or good to eat, as favorable terms which reassure the ego and set it up as being socially desirable. He has also grades of defamation, which are identified with poisons and with excreta. These generally,

though not always, mean that the person is identified with the socially outcast. The well-being of the ego is determined by getting plenty of compliments every day from family and neighbors. Every one practices using both sets of identifications. The words used apparently are not particularly significant; it is the tone of voice and the affect with which the things are said that count.

I should like to discuss further this extremely interesting phase of human behavior, but my time limits me to calling attention to the significance of adherence to cloacal interests in children and of the tendency of persons who have been frustrated in society to regress by learning to think of themselves in terms of derogatory, anal-erotic defamations. If one calls a person vile names, and he is not a strong character, he will visualize himself in such terms and will then undergo a great disturbance of affect and physiologic function and a tendency to regression to a lower attitude. Such a condition can become very serious. Take, for instance, the patient with hebephrenic schizophrenia; such a person experiences defeat in the right to live in the heterosexual direction and, finally, a breakdown, with regression to cloacal affects and attitudes. This is largely tied up not so much with constitutional deficiency, for many of these patients recover, as with the pressure of the social group. Treatment often is successful if the patient can be made to think of himself again in better terms, to visualize himself in better terms, such as are pleasant to every one trying to get along in society and be successful. I am sorry that I cannot elaborate further on this extremely interesting paper, but it does perhaps make the point clear that the physician should give importance to the ontogenetic recapitulation of phylogenetic patterns from the cloacal phase all through the genital development.

Phenomena of Sensory Suppression. MAJOR NORMAN REIDER, Medical Corps, Army of the United States (by invitation).

This paper was published in full in the June 1946 issue of the ARCHIVES, page 583.

DISCUSSION

DR. S. BERNARD WORTIS: One can add little to the excellent paper Major Reider has given us. This more careful psychologic testing is being used with greater frequency in studying neurologic illness. With such organic lesions of the brain one sees psychologic and somatic factors delicately intertwined. Studies like this emphasize the importance of enlarging the methods and scope of testing in order to pick up these most subtle defects in function. On the neurophysiologic side these data on cortical suppression or extinction have come from the work of Dusser de Barenne, Garrol and McCulloch; and from the clinical side many of these disturbances in function have been described by Goldstein, Schilder, Riddoch, Morris Bender and Furlow. The physiologists have furnished data to indicate that certain areas of the sensory cortex in certain animals, chiefly monkeys, especially areas 4S, 3S, 2S, 8S and 19S, have suppressor strips, and therefore suppressor strip functions, and these can be related to the motor mechanisms. More recently clinicians with war experience have indicated that these reactions are seen in cases of cerebral trauma and that with adequate testing such defects can be elicited. Many a disturbance in function of which soldiers with cerebral injuries complain, although on the surface appearing as a neurotic manifestation, has as a basis injury to cortical areas and gives evidence of definite pathologic localization of the lesion. Moreover, Major Reider has emphasized the homolateral suppressor effect.

The author is to be congratulated on his clinical observations, which further elucidate the psychologic, physiologic and functional factors of cerebral function.

DR. E. D. FRIEDMAN: May I ask Major Reider whether he considers these sensory suppression phenomena allied to Babinski's anosognosia?

MAJOR NORMAN REIDER: Certainly, it is likely that they are. I think Dr. Bender's recent paper (Extinction and Precipitation of Cutaneous Sensations, ARCH. NEUROL. & PSYCHIAT. 54:1 [July] 1945) touches on that point.

CHICAGO NEUROLOGICAL SOCIETY

Ben W. Lichtenstein, M.D., *President, in the Chair**Regular Meeting, Dec. 11, 1945.***Paroxysmal Attacks of Unilateral Sweating Associated with a Pontile Lesion: Report of a Case.** DR. JOSEPH A. LUHAN.

A Negro aged 23 entered Cook County Hospital complaining of a "dead feeling" in the right extremities, blurring of vision and attacks of sweating on the right side of the body associated with dizziness. He had been subject to nocturnal convulsive seizures since the age of 4 years.

About a month before admission, while at work during the day, he suddenly became dizzy and broke out in profuse perspiration on the right side of the body from the head to the toes, inclusive. He went to the washroom to sit down and found that he was unable to wipe the area dry because of profuse sweating. In about twenty minutes the sweating and dizziness stopped. He had had many spells of sweating and dizziness since the first attack, without any of his former "sleeping spells."

Examination on admission disclosed marked diminution of all forms of sensibility on the right half of the body (including the face), right hemiparesis and palsy of the left sixth nerve. He was seen in a number of attacks of profuse sweating on the right side. A roentgenogram of the chest revealed bilateral infraclavicular infiltration suggestive of tuberculosis. Subsequently, there developed conjugate deviation of the eyes to the right; then weakness of the masseter muscle, pronounced deafness and, finally, facial palsy of peripheral type appeared on the left side. The patient died eighty-one days after admission.

Necropsy revealed a solitary tuberculoma confined to the caudal four fifths of the pons, without generalized meningitis; the lesion was secondary to pulmonary tuberculosis.

This case is of interest if for no other reason than the factual observation that paroxysmal hemihyperhidrosis can occur. The patient had had so-called idiopathic epilepsy prior to the development of these attacks of sweating, which were in some way associated with an expanding focal lesion in the left side of the pons, starting probably in the vicinity of the medial lemniscus at about the level of the sixth nerve. It is probable that most of the pontile tegmentum on the right side was intact when the attacks of sweating began.

Neurologic Disturbances Associated with Multiple Myeloma. DR. LEROY H. SLOAN, DR. R. W. KEETON and DR. LOUIS LIMARZI.

Twenty-one cases of multiple myeloma were reported, 11 from the Illinois Central Hospital and 10 from the Research and Educational Hospitals. In 17 of this number the diagnosis had been confirmed by sternal puncture. In 2 cases there was a single isolated myeloma, with a sciatic syndrome in one and a slowly developing syndrome of compression of the cord in the other; in the other cases the lesions were multiple.

The chief complaint was backache; the pain was progressive and severe, radiated anteriorly and was increased by effort, particularly during the act of turning in bed, as well as by coughing, bearing down and local pressure. All levels of the spinal vertebrae were involved. The essential neurologic syndrome was that of compression of the cord, with development of flaccid or spastic paraplegia or quadriplegia, depending on the level of invasion. No isolated cranial nerve palsy was observed. The optic disks showed no evidence of increased intracranial pressure. In 1 case there was deafness due to local conditions not associated with the myelomatous involvement. Convulsive seizures did not occur, even in the presence of extensive destructive changes in the skull. Ascending cystopyelonephritis was the usual cause of death; in several instances true uremia developed.

Laminectomy was of temporary benefit. Roentgen irradiation was the usual treatment for the local process, and blood transfusions were given to combat the anemia which developed as myeloma cells invaded the bone marrow.

Sternal puncture is of maximum importance in diagnosis of lesions of the bony framework, especially when associated with compression of the spinal cord. In 2 cases in this series biopsy completed the diagnosis, but sternal puncture is more valuable when the fluid is examined by one cognizant of cell types and changes. Diffuse areas of rarefaction in bone calls for sternal puncture unless one is certain that a localized primary tumor explains the rarefaction as a metastatic lesion. The diagnosis of localized tumor formation in the spinal vertebrae may be aided greatly by this procedure, even though the tumor appears to be of malignant origin rather than part of an undisclosed multiple, diffuse process. A roentgenogram of the skull will also be of aid, since in multiple myeloma the skull is usually invaded and the characteristic punched-out areas appear; these areas may appear earlier in the mandible than elsewhere in the skull.

The chemical features of multiple myeloma are hyperproteinemia, reversal of the albumin-globulin ratio, hyperglobulinemia and hypercalcemia with normal or high serum phosphorus. The blood picture is that of normocytic or macrocytic anemia, autohemagglutination, frequent leukopenia with atypical plasma cells and myeloma cells and the presence of myeloma cells in the bone marrow. The absence of such cells does not rule out the possibility of multiple myeloma. The neurologic picture is due in most instances to compression of the spinal cord and regional roots, with radiating pain, the pain being made worse on movement, and is associated with gradual development, for example, of paraplegia, quadriplegia, loss of bowel and bladder control.

DISCUSSION

DR. PERCIVAL BAILEY: I was most impressed with the high incidence of involvement of the spinal cord and the usefulness of spinal puncture in diagnosis. Sometimes it is difficult to make a differentiation, roentgenologically or clinically, of metastatic disease, Hodgkin's disease and myeloma.

DR. L. LIMARZI: One of the telltale signs in cases of multiple myeloma is the extremely soft, nonresistant or "cheeselike" consistency of the sternum that is encountered while the needle is being inserted into the sternum for aspiration of marrow for biopsy. This characteristic consistency of the sternum should always make one aware of or suspect multiple myeloma.

DR. LEROY H. SLOAN: In the syndrome of compression of the spinal cord one must, of course, consider the likelihood of myeloma. Diagnosis will be greatly aided by sternal puncture and by roentgenograms of the skull and mandible, as well as by the finding of hyperproteinemia with high globulin content and reversal of the albumin-globulin ratio. I wish to thank Dr. Limarzi, who examined all the sternal fluids in this series.

Oneirophrenia, a Clinicophysiological Syndrome. DR. L. J. MEDUNA and DR. W. S. McCULLOCH.

The clinical syndrome called oneirophrenia consists in an oneiroid picture, as described by Ragis and by Gross, and a specific disorder of carbohydrate metabolism, indicated by a somewhat sustained level of the blood sugar in the usual glucose tolerance test and in the Exton-Rose glucose tolerance test and by resistance to insulin, due to some factor circulating in the blood and detectable in the urine by bioassay, as indicated in collaborative investigations by Gerty, Urse, Braceland, Vaichulis and their associates. The temporal relations of the psychiatric picture to the biochemical picture were discussed. These observations indicate that the carbohydrate metabolism is disordered during the psychosis and is normal during remissions, spontaneous or induced. There is even evidence that the biochemical changes, as detected in the blood stream, occur about one day before the changes

in clinical symptoms, both on the patient's going into and his coming out of the psychosis.

DISCUSSION

DR. FRANCIS J. GERTY: It is now known that the "shock" treatments do not give equally good results in all cases of "functional" and "constitutional" psychoses, even when therapy is begun early. Excellent results have been obtained when the initial diagnostic classification has been in the schizophrenic, manic-depressive or unclassified group. If prior to the use of sedation the patient has had evidence of confusion, a better prognosis is generally offered. The use of excessive amounts of sedatives and the effects of near exhaustion from hyperactivity and loss of sleep may interfere somewhat with making a correct judgment as to whether the confusion is a primary or a secondary symptom of the disorder. If the confusion is a primary symptom, one may justifiably believe that pathologic biochemical changes are present which result in a psychotic reaction resembling in some respects the symptoms one associates with schizophrenia, manic-depressive psychosis and other categories of the psychoses. It is in these cases, regardless of the superficial suggestion as to classification along old diagnostic lines, that recovery occurs. If the confusion is merely a secondary phenomenon or is not present at all, the results with shock treatment probably are no better than those without such treatment. If one looks only for the cruder evidences of extreme confusion, one will probably miss detection of many cases that belong in this group. It is unfortunate that there is no better means of detection of these cases than clinical estimate of the mental symptoms. These symptoms represent merely a reaction. Discovery of what lies behind the reaction is still the real problem.

Electronic Devices for Use in Modern Neuropsychiatry. MR. CRAIG GOODWIN.

To design apparatus for use in electrophysiology, the engineer must study the problem firsthand to learn what is required of the equipment. Successive revisions of the first design should result in apparatus whose controls are few, orthogonal and stepwise.

One stimulator developed thus uses a variable frequency master oscillator, giving a repetitive, condenser discharge type of stimulus. Other features are a variable coupling condenser, which provides control of wave form, i. e., of the time constant; a three decade voltage divider for control of output, and a degenerative power amplifier with a step-down output transformer. The output is thus of the low impedance, or "constant voltage," type, and its voltage does not vary in response to changes in load or electrical resistance of the preparation. This prevents apparent variations in threshold when pressure of the electrode or the area of contact changes. Finally, the output is obtained from the secondary winding and is thereby isolated from the chassis and from ground. This reduces artefacts and precludes accidental shocks.

In direct current amplifiers preference is given to the method of using a "voltage divider" coupling between stages. This requires a source of voltage above ground and one below. The latter permits the use of a large common cathode resistor to provide differential and push-pull actions, which are generally desirable in biologic amplifiers. The "voltage divider" coupling also makes possible an amplifier whose input and output are each nearly symmetric about ground, standardizing and facilitating interconnections between amplifiers. Thus, a universal low gain amplifier can be used, and two or more may be cascaded if necessary.

The final amplifier should be a single stage, multiple channel amplifier designed simply for the recorder used (crystal or magnetic ink writer or cathode ray, for example). This minimizes retirement of apparatus when recording methods are changed.

DISCUSSION

DR. JAMES G. GOLSETH: Mr. Goodwin has described several measuring instruments which may enable one to learn more about the nervous system. It is not important at this time whether one agrees or disagrees with Mr. Goodwin regarding the relative efficacy of a constant voltage, saw-tooth wave as compared with a constant current, square wave for cortical stimulation. It is of great importance, however, that both instruments measure fundamental physical quantities—the former, voltage and time; the latter, current and time. Given such instruments, the investigator may then find out for himself which type furnishes the more accurate data and, in particular, the more reproducible data. In other words, if he correctly employs reliable instruments which measure in a reproducible manner fundamental physical quantities, he may then let the data obtained speak for themselves.

MR. JAMES A. FIZZELL: For the past few years I have been doing work slightly analogous to that of Mr. Goodwin, but I do not feel that I am qualified to criticize his work. I am highly impressed with the various pieces of apparatus he has devised, for I can see and appreciate some of the difficulties he has overcome.

It began to appear ten or fifteen years ago that certain fields of medical research would be benefited if electronics could be applied. The field of neurology is a particularly rich one for such application, inasmuch as the nervous system is so strongly analogous to a large communications system. In the development of a modern telephone system, it was necessary to design and build elaborate pieces of test equipment. They were marvelous instruments, but they did not transmit telephone messages; they were only tools. The stimulator and the amplifiers developed by Mr. Goodwin are simply tools, regardless of how elaborate they may be.

The first instrument which Mr. Goodwin presented might be called a signal generator. It was said to have four desirable features. I am sure Mr. Goodwin will admit it has several more, but he has mentioned the principal ones. The amplifier is a test equipment, like a monitor set, which permits tapping in on a telephone line. Application of these pieces of apparatus to the study of the central and peripheral nervous systems is actually to apply them to the greatest of all communication systems.

An outstanding feature of this work was suggested when Mr. Goodwin stated that his initial efforts at solving a given problem usually proved to be merely a fixed approximation. His later success came about as a result of cooperation between him and the medical men with whom he worked. The remarkable feature of all these electrical tools is that they are the result of cooperation: the small and powerful diathermy machines, the portable electrocardiographs, the multiple channel electroencephalographs—all are beautiful evidences of cooperation between workers in two highly specialized sciences.

The electronics engineer did not know intuitively that an acceptable input current to an electroencephalographic amplifier would be 10^{-10} microamperes; he found that out by cooperation with medical men. The medical man did not know that reducing the heater temperature of the first stage would reduce emission velocity, noise and grid current while increasing the input resistance; he learned and profited by cooperation with the electronics engineer.

These electronic instruments are wonderful, but taken by themselves they mean very little, because they are simply tools. In order to make the best use of them and to make the greatest advance in neurologic research, it is necessary that workers who use them know something about them and be able to think in terms of them. This means a more complete merger of the two specialized fields of neurology and electronics, which can be accomplished only by considerable study and hard work. I believe that if members of the medical profession learn to use this apparatus correctly, develop an understanding of it and seek more uses for it, there will result a multiplication of such devices, thus speeding advance in neurologic research.

Book Reviews

The Diagnosis of Nervous Diseases. By Sir James Purves-Stewart. Ninth edition. Price, \$11. Pp. 880. Baltimore: Williams & Wilkins Company.

This is the ninth edition of an excellent work in neurology. The subject is studied from the practical viewpoint of the clinician, with the orientation toward symptoms and syndromes rather than disease entities.

This edition has brought up to date the subject of neurology, with greater emphasis and study on modern methods. The form, printing and illustrative diagrams and photographs are more numerous and discussions are more lengthy and detailed than in the previous editions.

In the preface the author comments on the impact that the recent war has had on neurology and psychiatry. A number of new neurologic syndromes have been revealed, such as the crush syndrome and the visual disturbances of aviators, to mention a few.

The first three chapters deal with physiologic anatomy, and there is an excellent section on methods of case taking. There are chapters on delirium, coma, convulsive disorders, involuntary movements, aphasia and disorders of articulation and a discussion of the cranial nerves.

There are two chapters on pain and other abnormalities of sensation, which are rather comprehensive. There follow chapters on organic paralyses of the upper and lower motor neuron types, recurrent and transient palsies, incoordination, posture and gait, trophoneuroses, reflexes and the vegetative nervous system.

The section on the psychoneuroses and psychoses suffers from the purely organic approach to mental illness and at many points becomes an essay, with sharp criticism of the psychoanalytic theory and method.

The chapters on electrodiagnosis and prognosis, the cerebrospinal fluid and encephalography, disorders of sleep and intracranial tumors are good. There are a useful index and provocative references.

Both elementary and advanced students of neurology will find this book a useful and practical text.

Our Inner Conflicts. By Karen Horney, M.D. Price, \$2.75. Pp. 250. New York: W. W. Norton & Company, Inc., 1945.

In this book, Dr. Horney continues the elaboration of her ideas of the nature and dynamics of the neurotic character structure, with their challenge to the orthodox, strictly freudian psychoanalytic concepts, which she elaborated in her previous works. This volume presents the thesis that "the conflict born of incompatible attitudes constitutes the core of neurosis and therefore deserves to be called basic. . . . It is the dynamic center from which neuroses emanate. . . . neuroses are an expression of a disturbance in human relationships."

The various methods and technics which neurotic persons use to attempt to "solve" this basic conflict are then delineated, with Horney's usual lucidity of description and penetration of clinical observation. She classifies these neurotic mechanisms into the following general groups: moving toward people, moving against people, moving away from people, the idealized image and externalization. A vivid picture of neurotic hopelessness as the vault of unresolved conflicts is drawn and a theory of its relationship to sadistic trends suggested.

There is one problem that this book, by its very nature, raises but does not answer, namely: What are the cause and the nature of the disturbance in human relationships that produces the basic conflict and then the neurotic character struc-

tures? This defect is also reflected in the section on the means by which cure is effected in therapy, which, though optimistic, appears vague and general, in contrast to the preciseness and sharpness of the preceding sections.

Shock Treatments and Other Somatic Procedures in Psychiatry. By L. B. Kalinowski and P. H. Hoch. Cloth. Price, \$4.50. Pp. 294. New York: Grune & Stratton, Inc., 1946.

Kalinowski and Hoch have prepared a concise formulation of the results of clinical experience with and experimental work on the shock treatments and other somatic procedures used in therapy. Insulin shock treatment and the convulsive therapies are discussed in detail with regard to organization of treatment; technic; indications and contraindications; complications; medical, psychiatric, psychologic, neurologic, electroencephalographic and neuropathologic observations; prognosis, and results. There follow short chapters on other pharmacologic and physical therapies and on prefrontal lobotomy. A review of theoretic considerations concludes the text.

From a thorough knowledge of the extensive literature and a wide experience in the field, the authors have prepared this authoritative, well organized, lucid discussion of the subject. Controversial matters are objectively presented, and the authors' position is briefly stated. For details the reader is referred to the extensive bibliography. The authors take the position that the shock therapies properly applied in time are effective in many cases of schizophrenia and in the affective psychoses. Adverse reports in the literature are critically evaluated; and detailed recording of the type of case material, technic and end results is urged in future work.

The authors, however, are frank to confess that "we are treating empirically disorders whose etiology is unknown with shock treatments whose action is also shrouded in mystery."

This volume should prove useful as an introduction to these therapies for students and physicians. It is a good reference work for psychiatrists who are advising or applying shock treatment.

The Biology of Schizophrenia. By Roy G. Hoskins. Pp. 191. Price, \$2.75. New York: W. W. Norton & Company, Inc., 1946.

The material in this book is a slightly amplified version of the Salmon Memorial Lectures delivered by Dr. Hoskins in 1945. In this book the author has summarized his viewpoint. It is essentially a call to the medical profession not to neglect the retort and microscope in the deluge of the facile and seductive psychoanalytic approaches to mental disease.

In the first section, the author traces the development of man from an isolated atom to his present integrated self in society—quite an optimistic undertaking for sixty-seven pages! No issue is taken with the author's statements, but one wonders what his purpose was in initiating a review of so vast a subject, which of necessity had to be brief and which for the most part is known to the average scientific reader. For example, he allots exactly five brief pages to a subject he heads "The Nature of Man."

In the second section, he describes schizophrenia, again in such a brief manner that it suffers from underdescription. The inability to "empathize" is the psychologic core of the schizophrenic process. However, the author believes that schizophrenia is definitely a disease entity and that the substratum is an organic one. With this view in mind, he has conducted many years of investigation, particularly on the endocrine system. His own work and the work of others are briefly summarized in the last section. Research has attacked the schizophrenic patient from the vestibular canals to his vitamin tolerance. Metabolism, circulatory differences and the endocrine glands, individually and collectively, have been studied. No conclusions of any significance have as yet been drawn. But

the author is exceedingly optimistic. He sincerely believes that the solution lies in a qualitative and quantitative refinement in methods of investigation.

The book suffers from brevity, albeit the promising nature of the titles. Schizophrenia in its biologic setting is but postulated. Its proof in this book goes begging. The volume is recommended as an important contribution to the subject.

Principles of Dynamic Psychiatry. By Jules H. Masserman, M.D. Pp. xix, plus 322. Price, \$4. Philadelphia: W. B. Saunders Company, 1946.

In this book, the second in a series of three (or more), Dr. Masserman intends to make clear to the student the mass of material rapidly accumulating which is being organized into what is known today as dynamic psychiatry. He succeeds well in the first part of the book, in which he discusses the development of behavioral theories, from the behaviorism of Watson to the psychoanalysis of Freud and the psychoanalysis as it is practiced today. The Gestalt conceptions, the Meyerian psychobiology, the Adlerian schools, the Horney group (albeit briefly dismissed), the Pavlovian reflexology and the descriptive formulations of Kraepelin and Bleuler, all are discussed and criticized. Dr. Masserman pays his respects to all of them but calmly drops small fragmentation bombs in their midst, picking up some of the pieces and emerging with his own "biodynamic formulations of behavior," which is his concept of the true dynamic psychiatry.

He proceeds by setting down a set of criteria which he believes psychiatry should attempt to fulfil if it wishes to be classed among the sciences. Therefrom spring his four principles of dynamic psychiatry: (1) principle of motivation, (2) principle of experimental interpretation and adaptation, (3) principle of deviation and substitution and (4) principle of conflict. But that is not all. Based on his experimentation (briefly summarized in this book but extensively presented in his volume "Behavior and Neurosis," published in 1943), he has evolved at least five corollaries to each principle.

The author says of his own formulations that they "fall far short of covering all the phenomena of behavior and their possible interrelationships." However, the reviewers believe that his biodynamic formulations are instructive, stimulating and, above all, provocative of further thought, study and, especially, experimentation. Without doubt, the author will welcome any changes or regrouping of his formulations provided such are based on biodynamic principles of study. If he does not have the answer now, he has certainly opened a new road to research in psychiatry, which he invites all to travel with him. The "principles" as presented in this volume are not as fully developed as one would like, and it is hoped that the author's "Practice of Dynamic Psychiatry," now in preparation, will supply the integration, making his formulation more dynamically applicable to clinical material.

A serious criticism of a book of this kind intended for students is that it suffers from the complicated language used. Words such as solipsism, eschatologic, paranoidic, epinosic, ecdemomania, ecdysiasm, eleutheromania, eutelegensis, gelasmus, hetairism, hyponoic, koinotropy, letheomania, misocainia, pleniloquence, pleonexia and ululation do not add to quick understanding of ideas. On the other hand, the case illustrations and the illustrative psychoanalysis of a neurotic personality are very useful in elucidating the text.

The next book in the series is anticipated, but a change in the semantic style is recommended.